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Chronic Septic Infection of the Uterus and Its Appendages.*

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A FEW introductory words to indicate the object and scope of this paper will perhaps not be out of place.

In what follows I have tried to give a comprehensive, though necessarily incomplete account of the progress and results of certain inflammatory processes as they affect the uterus, tubes and ovaries. The subject is a large one, but it seems best in the first instance to deal with it in all its aspects, in order to avoid the purely regional point of view. The changes produced in the uterus will be considered more in detail.

There is no great difference of opinion as to the clinical history of chronic septic inflammation of the genital tract, but the symptoms of the disease, especially in its later stages, require more careful study. Good work has been done on the pathology of chronic inflammation of the tubes and ovaries; but there are only a few scientific observations on chronic inflammation of the uterus, and we are bound to admit that many pathological points are not yet definitely settled. Further, the failure of many to grasp the important part that chronic uterine inflammation plays in gynæcological practice is responsible for much in the way of treatment that is illogical and pernicious.

In dealing with the various kinds of cases, it will be most convenient if we reverse the order in which the infective process develops and take the more serious cases first.

* Read before the Obstetrical Society of London, January 2nd, 1907.

Cases in which the inflammation involves the uterine appendages.

The large majority of these cases begin with an acute or subacute infection, but occasionally they are chronic from the first. The illness generally dates from a confinement or an abortion, but in rare cases it may follow some minor operation on the uterus, such as the use of the sound. In acute infections, death may supervene before pathological changes in the organs affected are well marked. In the less acute infections, inflammation is set up which spreads along the mucous surfaces and penetrates into the tissues of the organs; and changes are produced, which remain more or less permanent. The process starts with an endometritis, then the mucous membrane of the tube is affected, and this leads to a localized peritonitis. The tubes and ovaries are thickened and may contain pus. But in the class of cases under consideration there is merely obliteration of the abdominal ostium of the tube, with adhesion of the tube to the ovary. The inflammation of the pelvic peritoneum results in adhesions; the ovaries and tubes may be bound together, and there may be more or less intimate matting of these organs with intestines, omentum and peritoneum.

The physical signs in the later stages generally consist of a thickening, more or less definite, at the sides of the uterus or in the pouch of Douglas. Sometimes the uterus is bound down by adhesions, and in this way a displacement is produced. The kind of displacement depends on the part of the uterus involved. Adherent retroversion is produced when the fundus is bound down posteriorly; pathological anteversion when the lower portion of the uterus is pulled back; and anteflexion is caused by the contraction of adhesions which draw the middle portion of the uterus backwards while the fundus and cervix are free.

The symptoms in cases of this sort, when they have passed into the chronic stage, are mainly caused by the condition of the tubes and ovaries. As a rule, the general health is much affected. The patient is easily tired, backache is common and also dysmenorrhœa, and there is sterility. In the earlier stages, there may be menorrhagia or irregular and frequent menstruation, but, later on, the periods may become scanty. There is often a marked change in the shape of the abdomen. The patient complains of having lost her figure and the abdomen feels and looks distended, and the muscles are rigid.

Cases in which the uterus is chiefly involved—Chronic metritis and endometritis.

The affection may, in the first instance, be acute or sub-acute, and

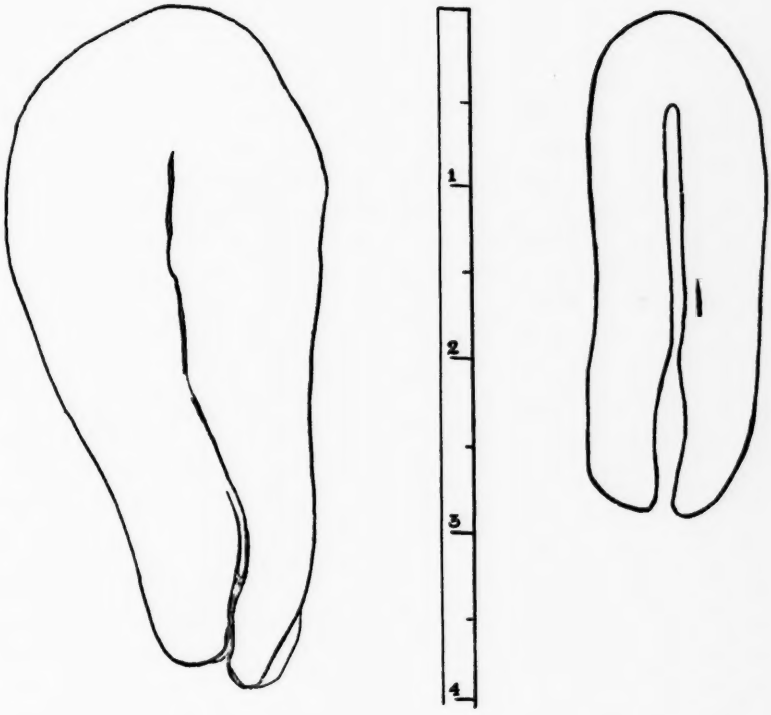

Fig. I. A diagrammatic comparison of a chronic metritic uterus with a normal uterus—natural size.

gradually become chronic. The uterus may be involved in an inflammation which spreads to the tubes and ovaries, and the inflammation in the appendages may clear up, leaving the uterus alone obviously affected. But in the majority of the cases the infection does not travel as far as the tubes, whilst it affects (directly or indirectly) the whole of the uterine tissue. The mischief is mainly uterine from the first, and there is never any acute stage. It is to this class that I wish particularly to direct attention.

Clinical history and symptoms. The trouble nearly always dates from a confinement or miscarriage, and the history is generally as follows:—

After the miscarriage or confinement the patient makes a recovery which may be classified as good, if careful observations of temperature and pulse are not made. But if the chart is kept strictly, variations from the normal, both in pulse and temperature, will show that convalescence has not run an ideal course. Whether these deviations from the normal are due to the same organisms which cause the more acute cases, but in smaller dosage, or whether they are due to some less noxious germs is doubtful; but it is certain that cases of what may be called "slight" or "less severe" sepsis are not uncommon.

As long as the patient is in bed she feels fairly well. It is only when she begins to move about that the symptoms show themselves. She then finds that she is easily tired, and she generally complains of pain, or it may be only of dragging or discomfort in the hypogastrium, or in one or both iliac regions. There is generally some leucorrhœa in the earlier stages, and when the periods are established they are apt to be profuse or long-continued. The lower part of the abdomen is sometimes distended. If the patient should become pregnant again, there is liability to miscarriage, and if this happens, the diseased condition is almost certain to be aggravated. In the later stages, the pain or dragging sensation in the iliac regions is common, and is aggravated by standing or walking. Leucorrhœa may persist for a long period but this is exceptional. Sometimes hæmorrhage becomes a prominent symptom, and this is especially the case in women over forty years of age. The hæmorrhage may be so profuse as seriously to affect the patient's general health and even endanger her life.

There is one symptom to which I should like to draw special attention, namely, the pain in the iliac region.

Pain or dragging in the iliac region is one of the commonest symptoms in gynæcological practice, and in my opinion is in the

large majority of cases caused by a heavy uterus. It is usually, but I think erroneously, classified as ovarian. If the bulk of the uterus is greatly increased the uterine body will fall either forwards or backwards, to a lower level in the pelvis than normal. This is bound to cause a pull or strain on the broad and round ligaments, and this pull or strain will be communicated to the side and front of the pelvis. Those who practise Alexander's operation rely on the round ligaments to pull the uterus up and keep it in a position of anteversion. It is reasonable to suppose that a heavy fundus, whether anteverted or retroverted, will pull on the round ligaments at their insertion into the abdominal wall. In cases of pelvic peritonitis—where the ovaries are involved in the inflammation—the most characteristic pain is in the back and not in front. But although in very pronounced retroversion or retroflexion the patient may complain of a feeling of pressure on the rectum or perineum, the iliac pain is much more common. It is customary, however, to refer pain in the iliac region in women to ovarian troubles, except in cases in which the appendix is thought to be at fault. In the discussion on this pain, which took place at the meeting of the British Medical Association in 1904, the uterus was hardly mentioned, and the conclusion is justified that the part played by the uterus in the production of this pain has been to a great extent overlooked. And yet it is the most constant symptom of a heavy uterus, whether that organ is retroverted or anteverted, as anyone may easily satisfy himself by the systematic interrogation of patients. It is aggravated or brought on by standing, and is generally most marked just before or at the onset of menstruation.

Physical signs. The one condition common to all these cases is enlargement of the body of the uterus. In some cases the cervix is lacerated and hypertrophied, or it is thickened without obvious laceration, or it shows an erosion. On bimanual examination, the body of the uterus is found distinctly and symmetrically enlarged, and it is tender to pressure. There is either retroversion or exaggerated anteversion, or if the isthmus of the uterus is unduly pliant, anteflexion or retroflexion is found. The appendages may be dragged down by the heavy fundus and are then easily felt, but they are not thickened or adherent.

Pathological appearances and etiology. Within recent years the operation of vaginal hysterectomy has been occasionally performed in cases of chronic metritis, and a study of the uteri thus obtained has widened our knowledge of the subject. In its naked-eye appearances the chronic metritic uterus differs widely from a normal uterus.

Fig. II. Chronic metritic uterus (natural size) removed from a patient aged 43, who had 5 children and 2 miscarriages. The symptoms were profuse menorrhagia, metrorrhagia and aching in the iliac regions. These symptoms were present for some years, but were more pronounced during the last 12 months. Patient was curetted about a year ago with only temporary benefit.

Fig. III. Another aspect of uterus shown in Fig II. Note the thickness of the endometrium.

In well-marked specimens the uterus weighs about three times as much as the normal uterus, its walls are about twice as thick, and there is great increase in all the dimensions (Figs. 1 and 2). When divided, the cut surface of uterine tissue bulges as if it had been under strain. In nearly every case the endometrium is considerably increased in thickness (Figs. 3, 4 and 5). There is no difference of opinion on these points amongst those who have been working at the subject, except as to the endometrium. Theilhaber believes that thickening of the endometrium is exceptional in chronic metritis—but Shaw, who has carefully examined a much larger number of specimens, found definite thickening of the mucosa in nearly every case.

When we come to microscopic changes the divergence of opinion is more marked. Some writers state that the most characteristic changes in chronic metritis are to be found in the blood vessels—others think that the connective tissue is most affected, while others regard the condition as primarily due to an increase in the muscular element. The fullest account of the microscopic changes in chronic metritis and the one which is based on the most copious material and careful investigation is to be found in a paper by Dr. W. F. Shaw, published in this same number of the Journal. I may here just mention briefly a few of Dr. Shaw's conclusions.

He attributes the increased thickness of the uterine walls to overgrowth of both muscle and cellular tissue—which in his view are increased almost equally, but with a very slight excess of connective tissue. He finds no constant changes in the vessels. The endometrium was definitely thickened in every one of twenty-five cases in which it could be measured, with two exceptions, both of which are easily explained.

There are three main theories as to the causation of chronic metritis. It has been regarded as due to (1) passive congestion; (2) inflammatory change; and (3) muscular hypertrophy.

The theory of *passive congestion* as a cause of chronic metritis may be stated thus: Under certain circumstances the venous circulation in the uterus is obstructed, and this obstruction leads to dilatation of capillaries and blood vessels, and possibly to certain changes in the walls of these blood vessels. An infiltration of small cells occurs in the neighbourhood of the vessels, and this passes into cellular tissue so that a permanent enlargement of the uterus is thus produced.

When we come to enquire into the conditions which are supposed to lead to this passive congestion we find that we are practically

limited to backward displacement of the uterus. Other conditions such as pelvic tumours, may theoretically cause chronic congestion of the uterus, but the uterine condition is then only of secondary importance. In certain cases of chronic metritis we find the uterus in a position of retroversion or retroflexion, and it is believed by some that these positions cause passive congestion, which in turn leads to permanent changes in the tissues of the uterus. Even if this were true it would not account for the larger number of cases of chronic metritis in which the uterus is anteverted, as this, the normal, position, even when exaggerated, cannot cause obstruction to the free blood return from the uterus. But there are reasons for believing that a mere alteration in the position of a healthy uterus does not cause passive congestion. The arrangement of the circulation in the uterus, as was pointed out by Sir John Williams * many years ago, makes it exceedingly improbable that this should occur. Then, the uterus is not always enlarged in cases even of very marked displacement; there may be very pronounced and long-standing prolapse of a uterus which is normal in size, if we except the tensile elongation of the supra-vaginal cervix, and yet this is the case of all others in which we should expect the blood return to be hampered owing to the stretching of the vessels. We find also now and again a uterus which has been for a long time in a position of retroversion, but which is not enlarged. Finally, if passive congestion is the cause of chronic metritis we should expect to find some well-marked and characteristic changes in the blood vessels of uteri which had long been retroverted and were enlarged. No such changes, however, have been proved to exist; on the other hand, Dr. Shaw's sections show that the changes in the vessels are variable and do not support any such hypothesis. The results of treatment give us another argument against the passive congestion theory, but this will be referred to when the treatment of chronic metritis is discussed.

In spite of all this, the theory of passive congestion has been widely, though for the most part tacitly, adopted, and it forms the basis of most of the treatment by pessaries and fixation operations.

There are writers who believe that the changes found in some cases of chronic metritis are to be found chiefly in the blood vessels, apart from passive congestion, but as I have stated, this cannot be accepted as a constant or even as a common change.

When we come to consider the remaining theories—the theory of *connective tissue inflammation*, and the theory of *muscular hyper-*

**Trans. of the Obstet. Soc. of London*, vol. xxvii. for 1885, pp. 112—117.

trophy—we have to admit that at present the position is not absolutely clear. Both theories are "a priori" possible, and each has been supported by observation. According to the one, the change is to be regarded as purely inflammatory; according to the other, it is—in part at least—in the nature of a true hypertrophy. This "working hypertrophy" may be produced by an increase in thickness of the uterine mucosa or by an increase in the amount of connective tissue between the muscle bundles. I believe that most cases of chronic metritis, if carefully examined, will show both hypertrophy of muscular tissue and increase of connective tissue.

It seems difficult to explain the divergent views of those who have written on the pathology of chronic metritis; as at first sight it seems that the question ought to be definitely settled by the careful study of microscopic sections. It may be that the cases from which specimens have been obtained differ widely in their clinical history; and that those who hold exclusively to the connective tissue theory have only examined specimens which have been obtained from uteri which have at some time been the seat of a very acute inflammation, or in which senile change is advanced. At the present time the microscopic anatomy must be regarded as "sub judice." I would suggest that in further investigations, the clinical history of each case should be carefully considered. The neglect of this can only lead to confusion. The term "metritis" may be a misnomer for some of the cases which are grouped under this title. An investigation into the pathology of the usual form of chronic metritis—that is, the type in which the uterus is enlarged and the symptoms have never been acute—is not made easier by including cases in which there has been acute inflammation after labour, or cases which have had a high temperature and a sharp illness after curetting, or cases of so-called climacteric hæmorrhage. Further, it is highly important that the whole thickness of the body of the uterus should be most carefully examined, and not one portion merely. Four types of case may at present be distinguished clinically:—1 and 2. The two types dealt with in this paper, *i.e.*, those which date from a confinement or abortion and are either simple or complicated by inflammation of the appendages. 3. The large uterus which bleeds profusely at the climacteric period (this may ultimately prove to belong to one of the former categories). 4. Uniform enlargement of the virginal uterus, in which the usual sources of infection can be excluded.

Another point that calls for careful investigation in these cases of chronic metritis is the condition of the endometrium. At present we are rather in the dark as to the relative importance of the changes in

the endometrium and those in the mesometrium in the production of symptoms. This is especially so in cases of profuse and intractable hæmorrhage. The theory which is involved in the word "fibrosis," and which assumes that the muscular tissue of the uterus is replaced by a fibrous tissue, and that the hæmorrhage is due to want of muscular control is an attractive hypothesis, but it has yet to be proved.

The *diagnosis* of chronic metritis is arrived at through the clinical history and the physical signs. The one point which I wish to emphasize here, is that chronic metritis in different stages and in varying degree is probably the most common of all the minor gynæcological ailments.

Treatment. If the opinions which I have expressed as to the pathology of chronic metritis are correct, it follows that all attempts to cure the condition by a mere alteration of the position of the uterus are quite illogical. The use of pessaries and the slinging up of the uterus by means of operation in cases of backward displacement of a mobile uterus are a tacit acceptance of the passive congestion theory. Some who use pessaries for such cases admit that they do not hope in this way to cure the patient, but merely to palliate her troubles. My own view on this point is that even where relief appears to follow, it is merely a kind of faith-healing, and that the use of pessaries for backward displacement eventually causes more discomfort than relief.

Others believe, however, that the maintenance of the uterus in what they call the "proper" position brings about a cure. The mere hanging up of the uterus by a round-ligament or fixation operation can never cause a reduction in its size, even if it succeeds in diminishing or altering the dragging symptoms.

The backward displacement of an otherwise healthy and normal uterus produces no symptoms of any consequence, and neither pessaries nor operations are required. When the uterus is enlarged by chronic metritis, the result to be aimed at in the treatment is the reduction of the weight of the uterus, and the cure of the tenderness, hæmorrhage, leucorrhœa, and dysmenorrhœa, if these are present.

The means at our disposal are comparatively few. Something can be done in the early stages by palliative treatment, rest in bed, frequent vaginal douching with hot water, tonics—such as iron and strychnine,—and careful attention to the bowels. The only time at which these measures are likely to be of lasting benefit is soon after the trouble has begun. It must, however, be remembered that in many cases the departure from the normal is comparatively slight,

Fig. IV. Chronic metritic uterus (natural size) removed from a patient, aged 52, who had had 9 children, but no miscarriages. The symptoms were menorrhagia and metrorrhagia since last confinement, *19 years ago*. For the last few weeks the hæmorrhage had been almost continuous. Note the thickness of the endometrium.

Fig. V. Chronic metritic uterus (natural size) removed from a patient, aged 32, who had had 2 children, the younger being 3 years old. The symptoms were menorrhagia and metrorrhagia for many years (beginning before marriage). Patient was curetted in January 1904, with only temporary benefit, and again in August 1905, with again only temporary effect on the hæmorrhage. The endometrium is much thicker than normal. Although this uterus is smaller than those shown in Figs. III. and IV. the microscopic sections of the mesometrium are practically the same in all three specimens.

and that no further treatment is advisable beyond the occasional employment of palliative measures.

In cases where we find marked enlargement of the uterus with typical symptoms, curetting should be done. The statement that is often seen in books, that curetting is followed by a decrease in size of the uterus in cases of chronic metritis, is by no means a flight of imagination. I have accurately noted it in my own cases, and Dr. Shaw's investigations have definitely proved that it occurs in a large percentage. Accurate measurement is possible only with the cavity, but careful bimanual estimation leaves no doubt in my mind that the whole uterus is usually diminished. The result of this reduction in size often is to allow the retroverted uterus to return to the normal position, but even when this does not happen the tenderness and dragging are removed.

It is important that the operation should be carried out with the most rigid asepsis. My own practice is to dilate under an anæsthetic with finely graduated metal dilators, to use a flushing curette, and to pack the uterine cavity with sterilized gauze for twenty-four hours. If there is cervical hypertrophy, a wedge should be removed from each lip of the cervix, and, if there is a tendency to prolapse of the vaginal walls, a plastic operation on the posterior wall adds to the patient's comfort afterwards.

It sometimes happens that a single curetting is not sufficient, and that the operation has to be repeated before a good result is obtained. In rare cases repeated curettings fail to relieve the symptoms. Whether this is due to a further production of diseased endometrium from the remains of the old or whether it is to be explained by the condition of the mesometrium, is uncertain. My experience leads me to favour the former view, and the fact that these failures after curetting are comparatively rare points to its being the correct explanation.

For really bad cases in which there is prolonged hæmorrhage which repeated curettings have failed to cure, there is a general agreement that vaginal hysterectomy is justifiable.

For my part I think the operation ought to be extended to some cases in which hæmorrhage is not a very important feature. There are a good many women whose lives are rendered miserable by chronic metritis at a time when they ought to be most active. If, in a case of this sort, the uterus is found to be much enlarged and very hard, and everything points to its being functionally useless,

and no improvement is brought about by repeated curetting, it ought to be removed. Owing to improvements in technique the operation involves very little risk, immediate or remote.

Since the above article was written an important paper on the subject of chronic metritis has been published by Drs. Gardner and Goodall of Montreal (*Brit. Med. Journ.*, November 3rd, 1906). So much care has been taken with the pathological material, and the paper gives such evidence of thoughtful study of the whole subject, that it is worthy of special reference. The authors give the clinical histories and pathological reports of nine cases. They evidently started with the impression that the terms fibrosis uteri and chronic metritis meant the same thing, as these terms are used as synonymous. In their summary, however, they show that in seven of the nine cases there was increase of the muscular tissue, and they emphasize the fact that in not one of the first six cases was the change a true fibrosis, but that there was hyperplasia of both the muscular and fibrous tissues. Case 7 was of an intermediate type, but 8 and 9 were cases of marked fibrosis of an adult type in which the vessels were involved in a true arterio-sclerosis. In both of these cases, however, it was easy to demonstrate arterio-sclerosis of the systemic circulation; one of them had a chronic interstitial nephritis and both showed cardiac hypertrophy: three signs indicative of senile change. One of these patients was aged 58 and the other 43.

It is a noteworthy point, that in not one of the 9 cases—with the possible exception of No. 2—is it possible on microscopical examination to classify the endometrium as normal.

These investigations of Gardner and Goodall's therefore tend to show that the most common type of chronic metritis is not a fibrosis but a hypertrophy; that fibrosis is exceptional and is probably only found in uteri which have undergone climacteric change. They show, further, that the microscopic changes which are usually found in the mesometrium in cases of chronic metritis are exactly the same as are to be found in sections of myomatous and fibro-myomatous uteri. The authors state that so similar are the sections from these two types of cases, that they find it impossible to distinguish them. They do not try to explain this similarity, but the reason seems fairly obvious. Both kinds of uteri are hypertrophic. In the case of the fibroid uterus it is reasonable to suppose that the hypertrophy is caused by the efforts of the uterus to expel the fibroid. In the case of the chronic metritic uterus the hypertrophy of the mesometrium,

in many cases at least, is probably secondary to hypertrophy, or some other abnormality—such as fibrosis—of the endometrium.

In this connection I would point out that some years ago I drew attention to the fact that the body of the uterus is often found to be hypertrophied in virgins, when the cervix is infantile in type with stenosis of the external or internal os (see this JOURNAL, April 1904). I brought forward evidence to show that this stenosis is often associated with hypertrophy of the endometrium. I had no pathological material at that time to show the microscopic nature of the change in the mesometrium, but I inferred that it must be a hypertrophy, as the double condition of the thickened mucous membrane and narrow canal was likely to lead to hypertrophy. Dr. Shaw has been able to show that chronic metritis in the virgin is indistinguishable microscopically from that which occurs in women who have had children.

The Pathology of Chronic Metritis.*

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So-called "chronic metritis" is a subject which has been clinically recognized and on which much has been written from early times, but it is only comparatively recently that its pathology has been discussed on anything like a scientific basis. Even now, vast as the literature of the subject is, there are hardly two authors who agree as to its causation and its pathology. The reason for these very different views probably lies in the fact that only in very recent years have uteri been extirpated for chronic metritis, the older writers having argued from post mortem uteri which had the same characteristics as those clinically diagnosed as chronic metritis, viz., enlargement and increased hardness, but which had probably not given rise to any symptoms during life. Even since these uteri were extirpated the views of modern gynæcologists seem as much at variance as those of the older writers, the most likely explanation being, that all the writers, except Lorentz and Theilhaber, were recording the results of the examination of only a very small number of such uteri.

In this paper I give the results of the microscopical examination of 38 uteri extirpated for chronic metritis alone and of 7 extirpated for chronic metritis along with some other concurrent disease: 3 had carcinoma of the cervix, 2 had intra-mural fibroids, 1 had a tubo-ovarian abscess and 1 double ovarian disease. For comparison with these I examined 23 normal uteri from patients of various ages and the endometrium removed by curetting in 50 cases of endometritis, the chief object of the latter being a comparison of their clinical histories with those of the chronic metritic uteri. In the following paragraphs dealing with changes in the blood-vessels, endometrium and connective tissue the figures are derived solely from the 38 cases of uncomplicated chronic metritis.

Pieces were taken from several portions of each uterus, and sections cut in paraffin so that each one included the whole thickness

* Based upon a thesis for which a gold medal was awarded at the examination for the M.D. degree at the Victoria University, Manchester, and read before the Obstetrical Society of London, January 2nd, 1907.

Fig. I. Group of vessels from the uterus of a virgin aged 18 years. The muscular tissue between the vessels contains a considerable amount of fibrous tissue, shown black in the photograph. The section was stained with van Gieson.

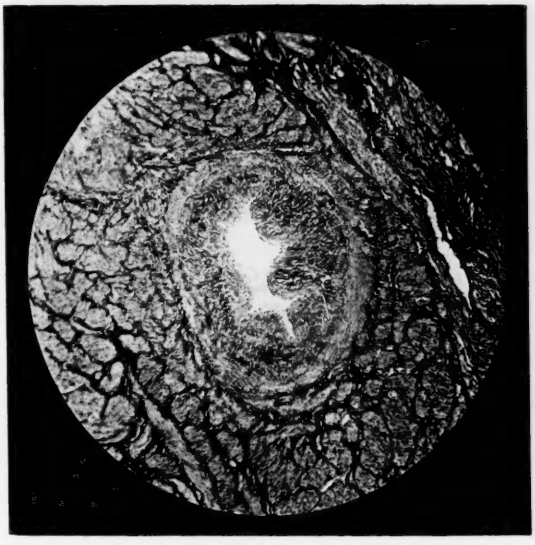

Fig. II. Vessel, from Case No. VI., showing great thickening of tunica media due to deposit of fibrous tissue, while the tunica intima and tunica adventitia are of normal thickness. There were only two vessels of this character in this section, the remainder being fairly normal (see Fig. III.). Section stained with van Gieson. This vessel is identical with those shown in Figs. IV. and V.

of the uterine wall from peritoneum to mucous membrane. These sections were stained with "van Gieson" as being the best differential stain for muscular and connective tissue, the connective tissue being bright red and the muscular tissue deep yellow.

Although differing on many minor points, most authors consider the pathology of chronic metritis to fall into one of two groups:—

(1) Changes in the vessels.

(2) Increase of connective tissue.

1. Changes in the Blood Vessels.

Several of the older writers considered the bleeding of chronic metritis to be due to changes in the vessel walls, which they describe as atheroma. Reinecke, in 1896, and Findley, in 1905, each published four cases in which they considered the hæmorrhage to be due to arterio-sclerosis, the thickening being chiefly in the tunica media.

Theilhaber found the vessels markedly increased in all his specimens, and the walls generally thickened, but he does not attach much importance to this.

I have investigated the condition of the blood-vessels in the 38 cases of chronic metritis, and compared their appearance with those of normal uteri at all ages, with the following results:—

Vessels not altered	15
A few vessels enlarged, but walls not thickened	2
Vessels decreased in number	6
Vessels increased in number	4
A few vessels with thick tunica media containing fibrous tissue	6
A few vessels undergoing hyaline or colloid degeneration	4
A combination of the last two	1
	38

In 21, that is 55 per cent., the vessels were not increased in number, nor were any changes observed in the vessel walls, but in the histories of these 21, hæmorrhage was of quite as frequent occurrence as in the other 17. In six instances some of the larger arteries showed great increase in thickness of the tunica media, the result of fibrous deposit. This is the pathological condition to which Reinecke, Findley, etc., ascribed the hæmorrhage of chronic metritis. In this I cannot agree, as I only found it occurring in a few vessels in six specimens out of 38 examined. Moreover, of these six

specimens, three (Nos. IV., X., and XVIII.) had no excessive hæmorrhage (see Fig. IV.), and I also found this change in the vessels of many multiparous normal uteri (see Fig. V.). In five instances the arteries showed a homogeneous degeneration affecting the media and adventitia. This area stained bright red with eosin, and pale yellow with "van Gieson." The exact nature of this change is not clear, but it is either a hyaline or colloid degeneration, probably colloid, as it stains a pale yellow with "van Gieson" (see Fig. VI.). I cannot, however, ascribe the abnormal bleeding of chronic metritis to this change, as the patients from whom three (Nos. XIV., XV. and XVIII.) of these five specimens were taken had no abnormal bleeding.

Anspach and Macgregor are the only authors I can find who mention this change. Anspach described it as a peri-arterial degeneration, which commences in the adventitia and finally leads to complete obliteration of the vessel. He does not name this degeneration, but states that it is a "hyaline coloured material" when stained with eosin, and bright yellow when stained with "van Gieson." Macgregor finds it in the vessels of the endometrium in uteri which show this degeneration in the vessels of the mesometrium. In none of my five specimens could I find a trace of it in the vessels of the endometrium.

From these results it seems clear that the hæmorrhage of chronic metritis does not depend upon changes in the vessel walls of the mesometrium.

The seven uteri extirpated for chronic metritis with some concurrent disease also showed little vessel change, in five of them the vessels were not altered, in one the number of vessels was increased but there were no other changes, while only one possessed vessels with thickened tunica media due to deposit of fibrous tissue.

2. Changes in the Relative Amounts of Connective Tissue and Muscular Tissue.

The most generally accepted view of the pathology of chronic metritis is that a great increase of connective tissue has taken place. Theilhaber considers that, not only has the amount of connective tissue greatly increased, but the amount of muscular tissue has actually decreased; this he believes to occur normally, as the menopause is reached and the excessive bleeding then commences owing to the loss of muscular control over the vessels unless these have proportionately contracted.

The method of estimating the amounts of connective and muscular

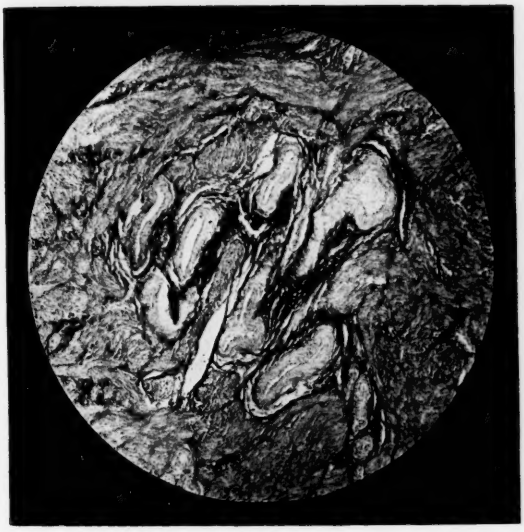

Fig. III. Group of normal vessels from the same section as Fig. II. (No. VI.). Stained with van Gieson.

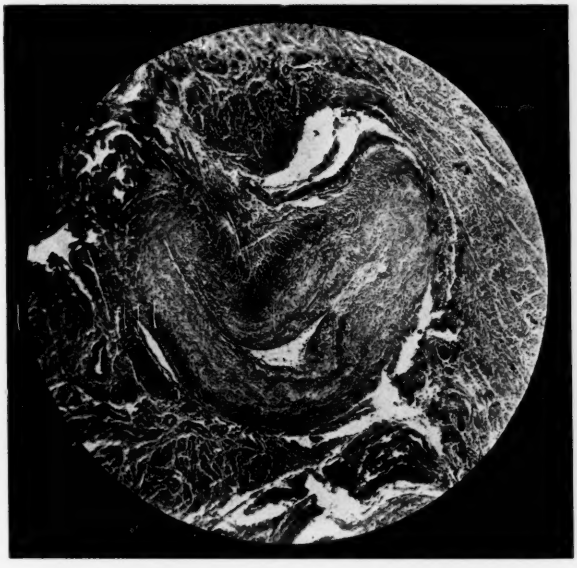

Fig. IV. Vessel showing thick tunica media due to deposit of fibrous tissue. From Case No. XVIII.; a uterus which had no abnormal bleeding. Stained with hæmatoxylin and eosin. This vessel is identical with those shown in Figs. II. and V.

tissue was as follows, and was similar to that employed by Meier, although I had been using it sometime before I read his paper.

The slide was put on a mechanical stage, under the " high power " of a microscope, and connective fields estimated through the whole length of the section, from peritoneum to endometrium; as these uteri were so thick this generally meant 40 to 50 calculations for each section. The mean of these estimates was taken as a fairly average estimation of that section. Each section was calculated two to three times, and the mean of these calculations taken as the final estimate for that section. Finally, if more than one piece had been cut from a uterus, the average of the results of the various sections was taken as the estimation for the uterus. In the later cases I only examined one piece, as I found very little difference in the several areas from the same uterus. Many of these calculations were made at intervals of several months, but, with few exceptions, the estimates for each section showed less than 5 per cent. of a difference, and very often less than 1 per cent. difference. In estimating a section I carefully avoided seeing the former results of that section, otherwise it would have been almost impossible not to have been influenced by the previous figures. The measurements of the thickness of the uterine wall were made by means of a mechanical stage on a microscope.

Forty-five uteri with chronic metritis were examined in this way, and also, for the sake of comparison, 23 uteri obtained post mortem from women of different ages, who had not complained of any uterine symptoms during life. Amongst these I was fortunate enough to obtain the uterus of a virgin, aged 18, who had died rapidly from the result of an injury to the head.

Meier has thoroughly worked out the musculature of the normal uterus in a series of 61 uteri taken at various ages, and has arranged them in a series of curves, showing the percentage of connective and muscular tissues at various ages. His first curve, containing all his uteri, shows anything but a regular gradation; his last, after eliminating those which are least likely to be normal, *i.e.*, puerperal uteri, and those of women who have suffered from wasting diseases, shows a regular increase of muscular tissue from childhood to about 20 years of age. For the next ten years the proportion of muscular tissue to connective tissue remains constant. After 30 years of age the muscular tissue progressively decreases in quantity until old age is reached.

I have not been able to obtain a sufficient number of adult normal uteri to arrange in a curve, but our results for corresponding uteri

are sufficiently close to show that our methods of calculating give very nearly the true proportion of muscular and connective tissue.

Meier divides his specimens into eight types, according to the arrangement of the connective tissue and muscle fibres. These groups can all be recognized in my sections if carefully looked for, but for practical purposes I believe that a division into four well-defined groups is all that is requisite:—

1. The connective tissue occurs as *dense, darkly-staining strands* between the muscle bundles; there is very little between the muscle fibres. See Fig. VII.

2. The connective tissue occurs as dense, *darkly-staining strands* between the muscle bundles, but, unlike Group 1, it also occurs in finer strands between the muscle fibres. See Fig. VIII.

3. The connective tissue is a *loose meshwork* between the muscle bundles, little connective tissue being present between the muscle fibres. See Fig. IX.

4. The connective tissue occurs as a *loose meshwork* between both the muscle bundles and the muscle fibres. See Fig. X.

The connective tissue is never equally distributed through the uterine wall; hence a section of one part must not be taken as indicating the proportion existing throughout the uterus. Thus the inner third of the uterine wall almost invariably shows a relatively small proportion of connective tissue to muscle.

Lorentz has thoroughly examined nine uteri, extirpated on account of excessive hæmorrhage, and with the diagnosis of chronic endometritis or chronic metritis. He describes the chief pathological changes as (1) a general increase in the size of the uterus; (2) increase in density of the uterus; (3) great increase of connective tissue; (4) thickening of the vessel walls. He usually finds no changes in the endometrium.

In the 45 uteri examined by me I found general enlargement of the uterus, the result of hypertrophy of both muscular and connective tissue elements in its wall. In a few cases only did the vessels exhibit marked changes. In every instance endometritis, as shown by increased thickness of the endometrium, was present in the 38 specimens of uncomplicated chronic metritis.

Theilhaber has maintained that the characteristic changes of chronic metritis are, a great increase of connective tissue and a diminished amount of muscular tissue. He bases this opinion on the results of observations by Meier for normal uteri, and Lorentz for uteri with chronic metritis. My observations do not confirm this view. The increase in size is due to a general hypertrophy of both

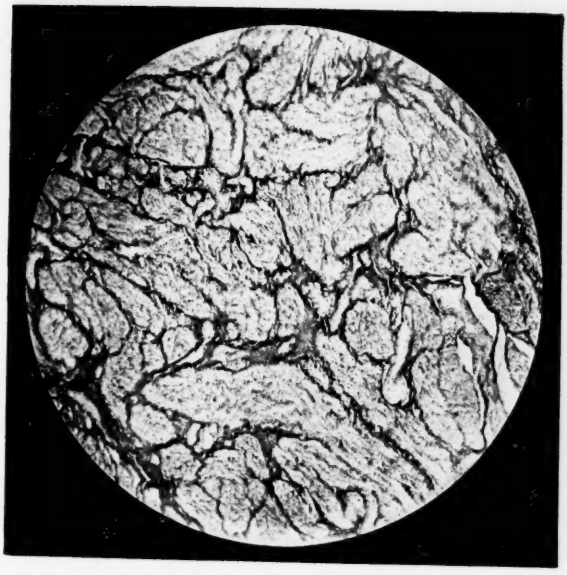

Fig. VII.

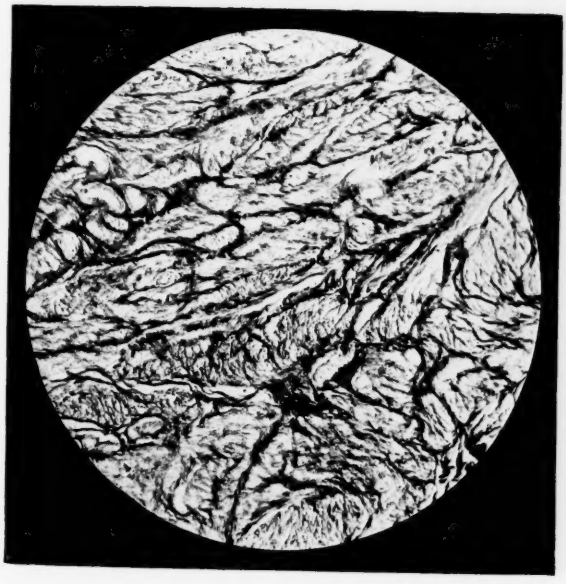

Fig. VIII.

Fig. IX.

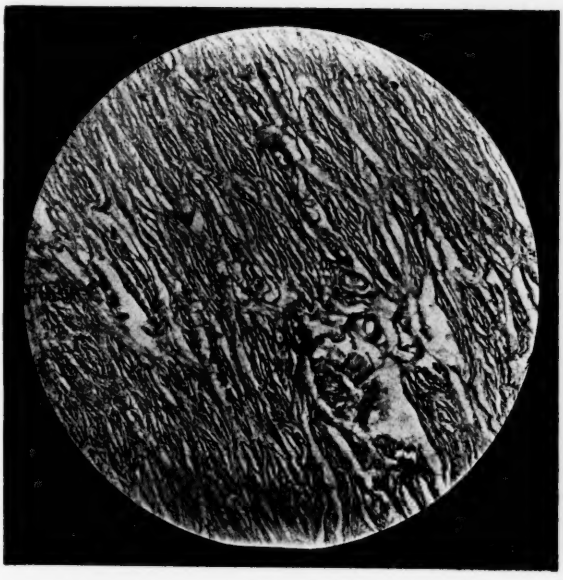

Fig. X.

elements, and there is certainly no diminution in the amount of muscular tissue.

Lorentz, in chronic metritis, places the proportion of connective tissue at 45 per cent. to 60 per cent.; average at 50·5 per cent. My results in similar specimens varied from 32·5 per cent. to 50 per cent.; average 40·4 per cent.

In the normal uteri of parous women Meier places the proportion of connective tissue at 46·5 per cent.; and my own observations show a ratio of 39·4 per cent.

It is thus clear that the difference between the proportion of connective tissue in normal uteri and in chronic metritis, amounting only to 4 per cent. in Meier and Lorentz's cases and 0·8 per cent. in mine, is too slight to account for the great increase in thickness of the uterine wall.

Increase in size and thickness of the uterine wall is a constant feature of chronic metritis. The dimensions of a normal uterus may be stated as follows (Quain):—Length, 7·5 cm.; breadth, 5 cm.; thickness, 2·5 cm.

All my chronic metritic uteri had much thickened walls, the least being 12 mm., the greatest 26 mm., and the average 18·1 mm.; while the average thickness of the walls of the normal uteri was only 8·7 mm. These figures were obtained after the sections were mounted, and are therefore below the correct measurement owing to the shrinking of the tissues in preparation; but as most of the uteri were prepared in the same way the difference between the average thickness of their walls will be about correct, or will err on the side of being too small. Now the chronic metritic uteri only show, on the average, an increase of 0·8 per cent. (in Meier's cases 4 per cent.) of connective tissue above the normal uteri, but their walls are on the average more than 100 per cent. thicker than those of the normal uteri. To say that this great increase in bulk is due entirely to increase of connective tissue, and that the musculature is actually lessened in amount, would be obviously incorrect. What has really taken place is a general hypertrophy of the whole uterine wall, the connective tissue having increased, in some specimens, in rather greater proportion than the muscular tissue.

Anspach, in his recent paper, also disagrees with these results of Theilhaber, his specimens of "metrorrhagia myopathica" showing no greater increase of connective tissue than multiparous uteri show with an equal number of pregnancies.

Several authors consider this "fibrosis" to be due to inflammatory changes commencing in the endometrium and extending to the mesometrium.

If this really were the case, we should then have to form two entirely separate groups, chronic metritis occurring in virgins, and chronic metritis occurring after pregnancy, as it is only in the latter that direct infection to cause the inflammation could occur. In only three or four of the uteri which I examined was there any trace of small cell-infiltration of the connective tissue; and in none was there anything to make us consider this the cause of the changes.

Condition of Mucous Membrane.

Theilhaber states that thickening of the mucous membrane is rarely found in chronic metritis, and when present is simply an œdema due to venous stasis. Lorentz found the glands increased in size or number in five out of nine specimens. In my 38 specimens I found it possible accurately to measure the thickness of the mucosa in only 31, since some of them had been badly preserved, or else only a small portion of the specimen was given to me which did not include the whole of the mucous membrane. However, in four others sufficient mucosa was left to show its characteristics, thus making 35 in all, from which definite conclusions could be drawn; in two the mucosa was of normal or diminished depth. Of these two, one (No. 15) had passed the menopause three years previously, and had had no discharge of blood since, only profuse leucorrhœa; the other (No. 24) had been curetted three times previously, and each time the mucosa had been found greatly thickened, but unfortunately I was not able to obtain sections from the previous curettings The 33 specimens had endometrium varying from 1·5 mm. to 5 mm. in thickness; but besides this increased thickness they all showed changes in the glands which distinguished them from the normal endometrium.

Unfortunately, almost every text-book gives a different classification of endometritis. Until the pathology of endometritis is thoroughly worked out and a standard classification adopted it is only possible to classify the various specimens according to their most prominent characteristics. I have divided the varieties of endometrium found in my specimens into six classes:—

1. The glands smaller than normal	4	specimens.
2. The glands enlarged	5	„
3. The glands increased in number	10	„
4. A combination of 2 and 3	10	„
5. The stroma increased	3	„
6. A combination of 4 and 5	3	„

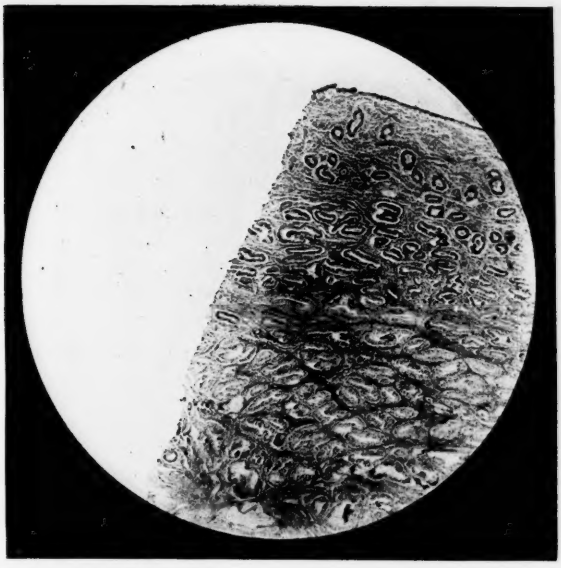

Fig. XI. Section of endometrium from No. XXXIII. The glands are enlarged and increased in number, the total thickness of the endometrium being much increased. (Compare with Fig. XII.)

Fig. XII. This shows the endometrium and part of the mesometrium from a normal primiparous uterus aged 32. This photograph is taken with the same magnification as Fig. XI., and shows how much the endometrium is thickened in cases of chronic metritis.

This allows the specimens to be classified according to their most prominent features; to do it more than roughly is impossible, as the various classes merge into each other; so much so is this the case that two sections hardly ever show exactly the same features. The density of the stroma varies very much, even in the same specimen, often being dense in the deeper portion of the section and loose in the superficial. In only eight specimens did I find the stroma less dense than normal, and in many it was denser; but even if the stroma were œdematous in every specimen, venous stasis would not explain the hyperplasia of glands found in so many instances.

These changes in the endometrium, along with the simple hypertrophy of the mesometrium, I find to be the only constant changes in uteri extirpated with the diagnosis " chronic metritis."

For the sake of comparison with these cases of chronic metritis I tabulated the symptoms (objective and subjective), and the result after curetting, of fifty cases diagnosed as chronic endometritis. The patients in both classes of cases complain of abnormal menstruation, intermenstrual pain, dysmenorrhœa and intermenstrual discharge; in both we find increase in the size of the uterus, flexion and tenderness. These symptoms certainly occur in varying proportions in the two classes of cases, but generally the difference is too slight to enable a definite diagnosis to be made. For practical purposes I find a patient is said to be suffering from chronic endometritis if her symptoms are recent and the uterus is not very much enlarged; if the symptoms are of long duration, if the uterus is much enlarged and hard, if hæmorrhage is the chief symptom, and especially if she has been curetted without relief, the diagnosis is chronic metritis. Chronic endometritis thus appears to be a precursor of chronic metritis.

That these symptoms are due to disease of the endometrium is proved by the excellent results of curetting, 39 (78 per cent.) of these 50 patients reporting at the end of six months that they were improved, and only 11 (22 per cent.) that they did not feel any better. Taking the individual symptoms separately, irregular menstruation, inter-menstrual discharge, pain and tenderness, the results were about the same, the effect on tenderness being the most marked. Of the very large number of these patients examined a few days after being curetted very few have complained of tenderness, although this was one of the most marked features of the examination previous to operation. At the end of six months only 8 per cent. of this series had any tenderness.

The statement is often made that the cavity of the uterus in cases

of chronic endometritis is lessened after curetting, but I do not know of any statistics on this subject. At the suggestion of Dr. Donald I measured the uteri of 23 women six months after they were curetted for chronic endometritis, and Dr. Donald has kindly given me two of his own private cases to bring the number up to 25. The cases were not picked in any way. I tried to re-measure every uterus whose measurement I had noted at the time of operation, but could not get more than 23 to come back again for examination; either they had moved to another address or were living too far away, or they "had not time to come."

TABLE SHOWING RESULTS OF CURETTING.

Number.	Age.	Social State.	Name.	Address.	Date of Operation and Length of Uterine Cavity.	Date of Second Measurement and Length of Cavity.	Difference.	Result to Patient.
					1905	1905		
lii.	27	M	C. W.	Lancaster	Feb. 8, 3½in.	Dec. 20, 2½in.	−1in.	Almost cured.
lix.	25	M	L. W.	Tyldesley	April 15, 3in.	Dec. 14, 2½in.	−½in.	Almost cured.
lxxvii.	25	S	N. E.	Bury	May 27, 3¼in.	Nov. 22, 3⅜in.	+⅛in.	No improvement.
xcvi.	26	S	M. B.	Altrincham	Aug. 2, 3in.	Dec. 17, 2½in.	−½in.	Much better.
xcii.	32	M	K. G.	Stockport	July 29, 3½in.	Dec. 14, 3¼in.	−¼in.	Greatly improved.
lxxxix.	35	M	S. N.	Golborne	July 26, 3¼in.	Dec. 14, 2⅜in.	−⅞in.	Greatly improved.
lxxxviii.	23	S	M. L.	Blackburn	July 19, 2⅞in.	Dec. 18, 2½in.	−½in.	No improvement.
xciii.	29	S	A. L.	Blackburn	July 29, 3½in.	Dec. 30, 3¼in.	−¼in.	Cured.
xciv.	23	S	E. C.	Cheadle	Aug. 2, 3¼in.	Dec. 19, 3in.	−¼in.	Great difficulty in passing sound; int. os almost occluded; patient improved
—	38	S	J. A.	Buxton	July 22, 2⅞in.	Jan. 4, 2⅜in.	−½in.	Better.
—	40	S	M. Y.	Patricroft	Mar. 16, 3¼in.	Dec. 13, 2⅞in.	−⅜in.	Much better.
xcviii.	29	M	F. W.	Irlams-o'-th'-Heights	July 31, 3½in.	Dec. 14, 3in.	−½in.	Much better.
—	29	M	E. C.	Middleton	Sept. 28, 3¼in.	Dec. 16, 3in.	−¼in.	Feels a new woman.
lv.	24	M	K. J.	Hulme	Mar. 24, 3¼in.	Nov. 10, 3in.	−¼in.	Better.
—	39	M	E. T.	Harpurhey	Mar. 14, 3¼in.	Dec. 15, 4in.	+¾in.	No better; int. os seems occluded; great difficulty in passing a sound.
lxii.	24	M	A. B.	Manchester	Apr. 28, 3½in,	Nov. 10, 3in.	−½in.	Much better.
lxxi.	35	M	E. M.	Hyde	May 19, 3in.	Dec. 11, 2½in.	−½in.	Almost cured.
lxxiii.	49	M	L. W.	Blackburn	May 23, 3¼in.	Dec. 5, 2⅜in.	−⅞in.	Improved.
—	28	M	E. H.	Whalley	May 23, 3in.	Dec. 5, 2½in.	−½in.	Not improved.
lxvi.	30	M	H. F.	Hightown	May 12, 3¼in.	Nov. 27, 2⅞in.	−⅜in.	Better.
xc.	30	M	E. F.	Knutsford	July 28, 3½in.	Dec. 15, 3in.	−½in.	Better.
xci.	22	S	C. B.	Heaton Chapel	July 28, 3½in.	Dec. 16, 2½in.	−1in.	Improved.
c.	31	M	E. R.	Bolton	July 17, 2⅞in.	Dec. 14, 3¼in.	+⅜in.	No better; great difficulty in passing sound through internal os.
					1903			
—	28	S	M. S.	—	Dec. 6, 3¼in.	Feb. 5, 2⅞in.	−⅜in.	—
					1904			
—	30	M	M.	—	Mar. 3, 3½in.	March, 2⅝in.	−⅞in.	—

In this list of 25, 22 (88 per cent.) showed diminution in length of cavity, and only 3 (12 per cent.) increase. The diminution varied from 1 in. to ¼ in., the average being ½ in. That this diminution has some definite bearing on the result is shown by the fact that the symptoms of the three which showed increase were not improved by the operation, while of the 22 which showed diminution of the cavity 21 were improved. Two of the uteri, which were increased in size, were very difficult to examine on account of the internal os being almost occluded, although the external os was patulous.

If the endometrium is hypertrophied from any cause the probability is that the uterus will endeavour to expel it, just as it does all foreign bodies; this would especially occur during menstruation, when the mucous membrane becomes more swollen, and would account for the severe pain which most of these patients complain of at that time. Any muscular body, with increased work, in the course of time hypertrophies. This is seen in the heart, stomach, bladder, etc., etc., and it is only reasonable to expect it in a uterus which has for some time been endeavouring to expel a hypertrophied mucous membrane. I have found this to be the case in all the specimens examined by me; certainly most of them showed a slightly increased percentage of connective tissue, but this was very small compared with the great total increase in the size of the uterus, an increase which is largely made up of new muscular tissue.

Theilhaber considers that the mesometrium is the starting point in so-called chronic metritis, and that the changes, if any, in the endometrium are only secondary. This, I consider, he deduces from insufficient data. He divides his patients with chronic metritis into six groups—(1) girls at puberty; (2) weak anæmic girls; (3) women at the menopause; (4) women with diseased tubes or ovaries; (5) women with myomata; and (6) women with subinvoluted uteri. He states that in these patients he finds atrophy of muscle and increase of connective tissue (although Lorentz's figures for chronic metritic uteri are not very different from Meier's for normal uteri, if averages are taken), and also increase of the size of the vessels. The uteri which he has examined microscopically and on which he bases his arguments, are those of Lorentz. Of these nine, seven were from women who had been pregnant from 3 to 13 times, and two were from married nulliparous women aged 32 and 38 years respectively. None of these can be taken as examples of his first two groups, viz., girls at puberty or weak anæmic girls. His third class, which includes women suffering from excessive hæmorrhage about the menopause also occurred in my series of cases; but I can find no evidence to

make me suppose that the symptoms are connected with the time of life, and are not the result of a primary endometritis, just as in all the other specimens.

Amongst the uteri which I examined, 14 were from women of 40 years old or more; of these, eight definitely dated the commencement of their symptoms to a confinement or a miscarriage; one had passed the menopause, and had had no hæmorrhage since; one had had the hæmorrhage for 9 years, commencing when she was 38 years old, and in another I could not obtain the date of onset of symptoms. This only leaves three (No. II., No. XX. and No. XXXIII.) which might possibly be due to primary changes in the mesometrium at the menopause, but, microscopically, I find appearances similar to those occurring in the other examples of chronic metritis; they show no specially high percentage of connective tissue, nor are the vessels enlarged or thick-walled, but both have a much thickened endometrium. Nor do Lorentz's specimens, except his first, appear to bear out this statement, the other two, of more than 40 years of age, having a pregnancy about the time of or after the commencement of the symptoms.

Out of 23 patients from whom I obtained a definite history, 14 dated the commencement of their symptoms previous to the last pregnancy, and only six had symptoms commencing at a considerable interval after the last delivery. This class, in which the symptoms date from a confinement or miscarriage (Theilhaber's Group No. 6), is the one into which I find most of these patients fall; I also find this borne out in the histories of large numbers of chronic endometritic patients. It is easy to imagine an endometritis being set up after a confinement or miscarriage, either through a slight degree of sepsis or from the retention of a small piece of membrane or placenta. This is sufficient to explain the symptoms (menorrhagia, metrorrhagia and leucorrhœa) of these patients; the dysmenorrhœa is explained by the uterus contracting and making an effort to expel the swollen endometrium during menstruation. In process of time the contractions lead to hypertrophy of the uterus, and so account for the enlarged, hard uteri and also for the constant aching pain found in these chronic metritic patients, due to the large heavy uterus dragging on the broad ligaments. That the endometrium is primarily at fault is proved by the results of curetting (see p. 133). In the great majority of cases, in a few days tenderness has disappeared and the uterus is smaller; at a later period the uterus is found to remain diminished in size, and the symptoms have disappeared or are improved. A certain number do not benefit from the first curetting,

and require a repetition of the operation; a very small proportion are not improved with repeated curetting, and in these hysterectomy may be necessary. Failure of the curette does not, however, prove that the endometrium was not primarily at fault, since a certain amount of endometrium must always remain behind, and may be the starting point of a fresh endometritis. In virgins, in whom endometritis develops without any obvious reason, the causal element has possibly not been removed and consequently may again develop endometritis. The specimens which I have been able to obtain, extirpated for chronic metritis, really represent the few failures in a very large number of patients on whom curetting was performed.

Chronic metritis may be associated with other pelvic or uterine diseases, such as tubo-ovarian inflammation, myomata and malignant disease of the cervix. I have examined seven examples of this association, two in which the metritis occurred along with small intramural fibroids, three with carcinoma of the cervix, one with a pyosalpinx, and one with double ovarian disease (? nature).

In each instance the uterus was much enlarged, globular in shape and increased in density, with the percentage of connective tissue slightly increased; in fact, the appearances, macroscopically, were identical with those of chronic metritis; microscopically, however, the endometrium was not increased in thickness.

These facts may be summarized as follows:—

Conclusions.

1. Chronic metritis is a simple hypertrophy of the mesometrium, and is not a connective tissue hyperplasia.

(*a*) The uterus in chronic metritis is much enlarged, the increase being in all diameters: Average length of uterus, 10·03 cm.; average length of cavity, 8·06 cm.; average width, 6·04 cm.; average thickness of wall, 2·48 cm.; average weight, 106·38 gr. The dimensions of a normal uterus are (Quain): Length, 7·5 cm.; length of cavity, 6·39 cm.; width, 5 cm.; the thickness of wall, 1·25 cm.; weight, 33·41 gr.

(*b*) The percentage of connective tissue varies considerably in the different specimens of chronic metritis, but many normal multiparous uteri possess an equal or even greater proportion of connective tissue; the average amount in the specimens of chronic metritis was only 0·8 per cent. higher than in the normal parous uteri.

(*c*) The vessel changes are variable, and similar affections are found in normal uteri.

(*d*) There is no evidence of active inflammation, as shown by small-celled infiltration or cicatricial tissue.

2. Chronic metritis is a slowly progressing affection of the uterus. In the early stage the uterus is simply enlarged, owing to hypertrophy of the mesometrium; at a later stage it tends to become more dense and hard, but this is not due to increase of connective tissue or to inflammatory changes.

3. Chronic metritis is never a primary affection. It is usually secondary to chronic endometritis, but may be associated with any pelvic or uterine disease leading to increased uterine contraction or vascularity, viz., fibroids, tubo-ovarian inflammation, carcinoma, prolapsus, etc.

4. The age of patients with chronic metritis varies from 23 years to 54, the average being 38·3. This is well below the average age at which the menopause occurs, and thus proves that the climacterium is not the causal factor in the complaint.

5. The symptoms are mainly those of the primary disease. In the early stage chronic metritis is indistinguishable from chronic endometritis. At a later period the great increase in size of the uterus may produce symptoms, i.e., sacralgia and aching pain in the hypogastric and iliac regions due to traction on the broad ligaments.

6. The diagnosis of chronic metritis is largely dependent on the result of treatment. In the early stage of the affection all the symptoms disappear after dilatation and curetting of the uterus, thus demonstrating that the endometritis is the primary cause.

7. In chronic metritis and chronic endometritis, local treatment of the endometrium may fail to relieve the symptoms. This is probably due to re-inauguration of the endometritis.

8. *Treatment.* A few patients are relieved by general treatment, *i.e.*, living a simple hygienic life with no very heavy work or too long standing, but with some occupation to prevent them from dwelling on their symptoms. The bowels must act freely; douching also seems to have a beneficial effect. The great majority of the cases require curetting, and are much improved by it (see p. 29). In a few instances the symptoms are not relieved even by repeated curettings; in these cases nothing short of hysterectomy is of any avail. This drastic measure is, however, only necessary in a small percentage of these patients.

At St. Mary's Hospital (Manchester), in 1905, 171 patients were curetted for chronic metritis or chronic endometritis, while in the same year only five hysterectomies were performed for these ailments.

For the large amount of material generously placed at my disposal I have to thank Dr. Lloyd Roberts, Sir Wm. J. Sinclair, Dr. Walter, Dr. Donald, Dr. Walls, and Dr. Arnold Lea. To Dr. Orr, of Prestwich Asylum, I have to tender my thanks for much kind help in preparing the micro-photographs.

Since writing the above paper a very important article has appeared by Drs. Gardner and Goodall (*Brit. Med. Journ.*, Nov. 3rd, 1906) on "Chronic Metritis and Arterio-sclerotic Uterus," based upon the examination of nine uteri extirpated for chronic metritis.

It is satisfactory to find that the description of the microscopic appearances which is given by the authors is substantially the same as that which I have recorded, although they arrive at very different conclusions. They found hypertrophy or hyperplasia of both muscular and fibrous tissues in seven out of these nine cases. Further, they note the similarity of sections of chronic metritic uteri, fibro-myomatous uteri and uteri with disease of the uterine adnexa. It will be remembered that in these last two kinds of cases I found similar changes in the uterine tissue, which I classified as "chronic metritis with some concurrent disease." So far we are in agreement.

In the remaining two specimens the authors found changes which, as they themselves admit, are not characteristic of simple chronic metritis, but which they regard as secondary to a general arterio-sclerosis. These changes I have not found in any of my specimens.

As regards their conclusions, they believe that the intractable hæmorrhage, which is so frequent a symptom, is due to muscular insufficiency of the uterine tissue and the vessel walls. They have arrived at this conclusion because the fibrous tissue of the uterus is slightly increased in comparison with the muscular tissue and because they found the adventitia of the vessels increased by a deposit of fibrous tissue in seven cases, and in some of the cases the media of the vessels was similarly affected. But, after most careful comparison of my sections, I have been able to establish no definite connection between these altered vessels and excessive hæmorrhage. Many of the specimens in which these vessel changes are well marked are normal uteri, while in other cases in which the hæmorrhage was most pronounced and long-continued, the vessels showed none of these changes.

In discussing the changes found in the endometrium the authors

state that they believe this affection to be primary and due to sepsis when it is found in what we may call the usual type of case, but secondary to arterial changes when it is found in their second or arterio-sclerotic type. How they differentiate between primary and secondary endometritis they do not state, nor is any difference obvious from their description of the microscopical appearances of their specimens. In my specimens of uncomplicated chronic metritis the endometrium was found thickened in all, with the exception of two which were easily explained, but in every case of uncomplicated chronic metritis the endometrium was of normal thickness or below it. But if changes in the endometrium were at any time secondary to changes in the mesometrium they ought to be found in the majority, at least, of cases of complicated chronic metritis. Further, the explanation of all cases of endometritis associated with chronic metritis as being due to either sepsis or secondary to arterial changes takes no account of the thickening of the endometrium associated with enlargement of the body of the uterus which is sometimes found in virgins (see Donald, *Jour. of Obst. and Gyn. Brit. Emp.*, 1904). Some of the specimens which I have examined were virginal uteri, with changes which could not be differentiated from those found in parous uteri.

Under treatment they only consider hysterectomy after preliminary curetting if the diagnosis is uncertain, thus evidently drawing a hard and fast line between chronic metritis and chronic endometritis. They also believe that ovarian function has some power over uterine hæmorrhage, and in support of this quote one case in which menorrhagia continued for some time after curetting, but underwent spontaneous cure in the course of a few months. In our experience this is what very often occurs after curetting, the patient generally receiving more benefit from the operation after a lapse of a few months than immediately subsequent to it.

Number.	Name. Age.	Social Condition	No. of Pregs.	Last Pregnancy	Symptoms Commenced.	Menstruation.	Menorrhagia. Metrorrhagia.	Inter-menstrual discharge.	Pain.	Clinical Examination.
i.	E.S. 44	M	3	7 yrs.	7 years	Regular every 4 weeks amount moderate	None	Watery and offensive	None	Uterus enlarged and fixed, anterior lip hypertrophied
ii.	G.A. 42	M	9	9 yrs.	2½ years	Periods last 3 weeks 1 week interval between	Menorrhagia and metrorrhagia	Leucorrhœa	Slight during menstruation	Uterus retroflexed, enlarged, tender, and fixed Left ovary tender, prolapsed
iii.	R. 35	M	2	10 yrs.	10 years	Regular every 28 days; duration 5 days; amount moderate	Menorrhagia after curetting	Profuse leucorrhœa	Severe dysmenorrhœa	Uterus retroflexed, mobile, enlarged
iv.	G. 42	M	1	12 yrs.	12 years	Regular scanty	Neither	Leucorrhœa	Constant aching in pelvis, back, and abdomen	Uterus enlarged, hard, antiflexed, and mobile
v.	Clinical notes not obtainable.									
vi.	G. 23	M	1	3 yrs.	3 years	Irregular not profuse	Neither	Leucorrhœa	Severe constant aching in pelvis; great dysmenorrhœa; dyspareunia	Uterus bulky, very sensitive, antiflexed Some perimetritis
vii.	H.G. 38	M	5	2 yrs.	3¼ years; after miscarriage	Menorrhagia and metrorrhagia	Both	Leucorrhœa	Pain in back and hypogastrium	
viii.	R. 26	M	1			Menorrhagia	Menorrhagia	Leucorrhœa	Constant pelvic pain dysmenorrhœa	Uterus retroflexed and tender
ix.	Clinical notes not obtainable.									

Macroscopical Condition.	Thickness and Description of Endometrium.	Thickness of Mesometrium.	% Connective Tissue.	% Muscular Tissue.	Blood Vessels.	Operation.	Clinical Diagnosis.
Weight = 82·415 gm. Length = 11·27 cm. Length of cavity = 9·85 cm. Thickness of wall = 2·5 cm.	3 m.m. Glands enlarged & very numerous in deeper layers Stroma dense in deeper part Vessels few and small	18 m.m.	37	63	Few in number & small. Walls not thickened and contain no fibrous tissue; very litle fibrous tissue about them	Vaginal hysterectomy July, 1904	Ch. metritis
	3 m.m. Glands enlarged and increased in number Stroma moderately loose Vessels increased in number	22 m.m.	43·8	56·2	Slightly above average number; vessels small and walls not thickened; no fibrous tissue in walls	Vaginal hysterectomy May, 1899	Retroversion Ch. metritis
Cavity = 8.25 cm.	3 m.m. Glands greatly inincreased in number Stroma loose Vessels average	21 m.m.	39·3	60·7	Smaller in number and size than normal; none enlarged, none with thickened walls; no fibrous tissue in walls	Curettage and ventral fixation; 6 months later vaginal hysterectomy	Ch. metritis
	3 m.m. Glands increased in number and size Stroma dense in deeper parts, loose in superficial Vessels very few	20 m.m.	41·3	58·7	About average number; a few increased in size, have thick walls, due to fibrous tissue in tunica media	Vaginal radical operation Sept., 1902	Ch. metritis
	3 m.m.—5 m.m. Epithelium raised into papillæ Glands enlarged, greatly increased in number Stroma fairly dense Vessels not enlarged	16 m.m.	38·7	61·3	Below average in number and size; walls not thickened; no fibrous tissue in tunica media		
	1·5 m.m. Glands slightly enlarged Stroma fairly dense and increased in quantity	15 m.m.	40·2	59·8	About avg. number; a few have very thick walls and narrow lumina	Ovarian abscess excised Nov., 1901 Vaginal hysterectomy June, 1902	Ch. metritis Ovarian abscess
	2 m.m. Glands enlarged, several veins dilated Stroma avg. density	16 m.m.	37	63	Increased in number; several have thickened walls, both intima and media; colloid degeneration in a few	3 curettings March, July, Oct., 1902 Vaginal hysterectomy March, 1903	
	3 m.m. Glands slightly enlarged, very numerous Stroma avg. density, small in amt. veins slightly dilated	13 m.m.	42·6	57·4	Fewer than average, not enlarged	Tubo-ovarian abscess removed Ventral fixation Sept., 1901 Vaginal hysterectomy Feb., 1902	Ch. metritis
	2 m.m. Glands enlarged and increased in number Stroma average Vessels average	22 m.m.	39·6	60·4	Rather above average number; a few vessels, in middle third, enlarged; walls not thickened; no fibrous tissue		

Number.	Name. Age.	Social Condition.	No. of Pregs.	Last Pregnancy.	Symptoms Commenced.	Menstruation.	Menorrhagia. Metrorrhagia.	Inter-menstrual Discharge.	Pain.	Clinical Examination.
x.	R.B. 37	M	4	5 yrs.	7 years	Irregular 3-5 weeks amount variable	Neither	Thick, yellow, and copious	Constant bearing down feeling left side Severe in left side & hypogastrium 1 day before and during period	Cervix lacerated Scar in left fornix of vagina
xi.	E.C. 52	M	13	13 yrs.		Every 14 days; duration 6-7 days; amount considerable	Both	Constant, copious, white discharge	In lower part of trunk 2-3 days before period	Vag. walls lax Cer. and os lacerated and eroded Uterus enlarged, fixed
xii.	H. 32	M	0		6 years	Every 28 days; duration 3-4 days	Menorrhagia	Profuse leucorrhœa	Severe in back and abdomen 2-3 days before and during flow	Os small and eroded Uterus antiflexed, mobile, enlarged Ovaries both thickened
xiii.	R. 37	M	1	8 yrs.	8 years	Every 21 days; duration 9-10 days	Menorrhagia	Constant leucorrhœa	Dysmenorrhœa	Cervix lacerated Uterus much enlarged, antiflexed, fixed Left tubo-ovarian mass
xiv.	M.B. 37	M	8	4 yrs.		Every 4 weeks; duration 3 days	Neither	Slight leucorrhœa	In back and around abdomen	Uterus large, round, mobile, not tender
xv.	H.W. 49	M	9	21 yrs.	8 years	Regular up to menopause 3 years ago	Neither	Yellow discharge for 8 months	None	Uterus pushed forward and fixed
xvi.	M.D. 37	M	10	6 mths	10 years	Every 1-2 weeks; lasts 7-8 days; amount considerable	Both	Constant brownish discharge	In back and hypogastrium 2-3 days before and during flow	Cervix torn into left fornix Uterus heavy, enlarged, firm, retroflexed
xvii.	W. 43	M	1	4½ yrs.	4½ years	Periods last 2-3 weeks	Both		In left side of abdomen	Cervix eroded and lacerated Uterus enlarged retroverted
xviii.	H.D. 35	M	12	1 yr.	1 year	Regular every 4 weeks average amount	Neither	None	Constant backache	Uterus enlarged and retroflexed

Macroscopical Condition.	Thickness and Condition of Endometrium.	Thickness of Meso-metrium.	% Con-nective Tissue.	% Mus-cular Tissue.	Blood Vessels.	Operation.	Clinical Diagnosis.
	2 m.m. Glands enlarged & increased in number Stroma increased and œdematous Veins dilated	14·3 m.m.	41·6	58·4	Some increased in size; a few have very thick walls containing fibrous tissue	Abscess incised left groin, 1898 Lt. ov. rmd. 1899 Dilated & cur. March, 1903 Vag. hyst. June 1903	Endometritis
Muc. mem. thick Uterus generally enlarged Walls soft and flabby Muc. polyp. 1·9 m.m. long	2 m.m. Glands dilated Stroma rather loose Vessels normal	20 m.m.	42·2	57·8	Some have small lumina and very thick walls, due to excessive fibrous tissue	Vaginal hysterectomy June, 1904	Ch. metritis
	2 m.m. Glands enlarged in deep layer, small in superficial Stroma varies Arterioles prominent in some patches	13 m.m.	39·7	60·3	About average number, not increased in size, nor have thickened walls	Oct. 1903, curet. Left tube and ovary removed Rt. ovy. resected Cur. Jan., 1904 Cur. Ap., 1904 Vag. hyst. Aug., 1904	
Cavity = 8·89 m.m.	Not obtainable	17 m.m.	39	61	Below average in number; a few with thickened walls due to fibrous tissue in middle coat	Vaginal hysterectomy April, 1901	
	2 m.m. Glands decreased in size and number Stroma increased and loose, arterioles enlarged and numerous	19 m.m.	48·2	51·8	Increased in number but not enlarged; walls not thickened, 1 or 2 show fibrous tissue and colloid or hyaline in walls	Curettage and iodine April, 1902 Vaginal hysterectomy July, 1902	Ch. metritis
	0·5 m.m. Glands decreased in size and number Vessels increased in number	13 m.m.	33·8	66·2	Increased in number; many enlarged, with thick walls undergoing colloid or hyaline degeneration	Vaginal hysterectomy	Ch. metritis
	3 m.m. Glands enlarged and greatly increased in number Vessels enlarged and numerous Stroma fairly dense	17 m.m.	41·9	58·1	Great increase in number; many enlarged, with thick walls showing colloid or hyaline degeneration	Curetting October, 1904 Vaginal hysterectomy November, 1904	
Length of uterus = 12·7 cm. Length of cavity = 10·16 cm. Width = 7·62 cm. Thickness of wall = 2·5 cm. Weight = 191·36 gm.	2 m.m. Glands rather more numerous and increased in size Stroma of average density Vessels more numerous, not enlarged	26 m.m.	39	61	Increased in number; size and walls normal	Emmet's June, 1891 Vaginal hysterectomy February, 1902	Ch. metritis
Length of uterus = 10·16 cm. Length of cavity = 7·62 cm. Width of uterus = 6·35 cm. Thickness of wall = 3·17 cm. Weight = 127·5 gm.	3 m.m. Glands increased in number and size Stroma varies Vessels about average number and size	26 m.m.	40·6	59·4	Average number; many enlarged, with thick walls showing much fibrous tissue; a few show colloid or hyaline degeneration		

Number.	Name. Age.	Social Condition	No. of Pregs.	Last Pregnancy.	Symptoms Commenced.	Menstruation.	Menorrhagia. Metrorrhagia.	Inter-menstrual discharge.	Pain.	Clinical Examination.
xix.	E. C. 40	M	4	1 year	1 year	Irregular	Both		Pain when flooding	Uterus large, retroverted, moveable, and tender Cervix lacerated Os patulous Appendages prolapsed
xx.	M. H. B. 54	M	2	27 yrs.	14 years	Irregular bleeding for 14 years	Both			Cervix small Uterus retroflexed, mobile
xxi.					Clinical Notes not obtainable.					
xxii.					Clinical Notes not obtainable.					
xxiii.	H. K. 36	M	11	16 mths	16 months	Prolonged heavy floodings	Both		Constant pain in left side; worse during menstruation	Cervix deeply lacerated Uterus globular, mobile enlarged Appendages normal
xxiv.	A. B. 30	S	0		6 years	Almost constant hæmorrhage for 6 years	Both		Slight dysmenorrhœa	Uterus enlarged, antiflexed, mobile, not tender
xxv.	E. V. 29	M	1	10 yrs.	2-3 years	Almost constant hæmorrhage	Both	Copious white	Constant aching in back; worse 2-3 days before and dur-menstruation	Os and Cerxix torn, eroded Uterus anteverted, antiflexed, enlarged Left ovary prolapsed, tender
xxvi.	K. W. 45	M	5	7 yrs.	7 years	Regular; average amount	Neither		Constant aching in back and abdomen	Uterus enlarged, retroflexed, fixed
xxvii.	? 37	S	0		9 months	Loses too much and too often	Both			

Macroscopical Condition.	Thickness and Description of Endometrium.	Thickness of Meso-metrium.	% Connective Tissue.	% Muscular Tissue.	Blood Vessels.	Operation.	Clinical Diagnosis.
	1·5 m.m. Not all present Glands increased in number, smaller in size Stroma dense Vessels average	24 m.m.	39·9	60·1	Increased in number; otherwise normal	Dilated and curetted September, 1904 Vaginal hysterectomy February, 1905	Chronic metritis
	2 m.m. Whole thickness not present Glands increased in number and size Stroma fairly normal Vessels fairly normal	19 m.m.	40·5	59·5	Inreased number; average size and no increase in thickness of walls	Curetted 1890 Curetted 1894 Vaginal hysterectomy 1904	Chronic metritis
	4 m.m. Glands increased in number, smaller in size Stroma loose Vessels few	18 m.m.	36·5	63·5	About average size and number		
Length of uterus = 8·89 cm. Length of cavity = 6·35 cm. Width of uterus = 6·35 cm. Thickness of wall = 2·54 cm.	5 m.m. Glands very numerous and slightly enlarged Stroma varies	21 m.m.	38·6	61·4	About average size and number		
	2 m.m. Whole thickness not present Glands much enlarged Stroma œdematous Veins dilated	21 m.m.	41·9	58·1	Average in number and size; no degeneration	Curettage and Emmet's July, 1899 Vaginal hysterectomy October, 1899	Chronic metritis
Cavity measured with sound = 7·62 m.m.	1 m.m. Glands increased in number and size Stroma dense	21 m.m.	37	63	Below average in size and number	Curetted 3 times Vaginal hysterectomy February, 1905	Chronic metritis
Length of uterus = 9·52 cm. Width at fundus = 5·08 cm. Thickness of walls = 2·29 cm. Weight = 70·87 cm.	1·5 m.m. Whole thickness not present Glands numerous and small Stroma dense Vesls. more num'r's Veins dilated	17 m.m.	46·7	53·3	About average in number and size	Curetted September, 1905 Vaginal hysterectomy June, 1905	Chronic metritis
	2 m.m. Glands greatly increased in number slightly in size Stroma loose Veins enlarged	12 m.m.	38·5	61·5	Average number and size	Vaginal hysterectomy July, 1905	Chronic metritis
Length of uterus = 8·89 cm. Length of cavity = 7·21 cm. Width at fundus = 4·44 cm. Thickness of walls = 1·67 cm. Weight = 53·014 cm.		19 m.m.	39·0	61·0	Rather below average in number and size	Vaginal hysterectomy October, 31, 1905	Chronic metritis

Number.	Name. Age.	Social Condition	No. of Preg.	Last Pregnancy	Symptoms commenced.	Menstruation.	Menorrhagia. Metrorrhagia.	Intermenstrual discharge.	Pain.	Clinical Examination.
xxviii.	A.C. 39	M	0		3 months	Regular and normal amount until 1905	Both		Dull aching in left iliac region since Dec., 1905	Uterus enlarged, antiflexed, and tender
xxix.	M.H. L. 47	M	4	13 yrs.	9 years		Both		None	Hard, irregular, retroflexed, fixed, about double normal size
xxx.	Clinical notes not obtainable.									
xxxi.	L. 43	M	5	18 mths	More than a year	7 days excessive flow with intervals of only one to two days	Both		Constant aching in iliac fossæ, worse during menstruation	Retroflexed, enlarged; not tender
xxxii.	M 32	M	2	3 yrs.	Always had menorrhagia, worse since last confinement and operations	Only free from hæmorrhage one week out of three	Both	None	None	Enlarged, not tender
xxxiii.	E.A. W. 44	M	10	3 yrs.	1 year ago		Both	Yellow offensive	Constant pain in lt. iliac region for two years	Cervix lacerated Uterus heavy and retroflexed
xxxiv.	Mrs. L. 40	M	3	8 yrs.	8 years ago	Menorrhagia 8 years much worse last 3 years Metrorrhagia 6 months	Both			Uterus large and retroverted
xxxv.	Clinical notes not obtainable.									
xxxvi.	A.B. 30	M	0		18 months	Menstruation excessive; sometimes too frequent	Both		Difficulty in micturition during menstruation	Uterus much enlarged, mobile

Macroscopical Condition.	Thickness and Description of Endometrium.	Thickness of Meso-metrium.	% Connective Tissue.	% Muscular Tissue.	Blood Vessels.	Operation.	Clinical Diagnosis.
Weight = 99·2 gm. Length = 10·16 gm. Length of cavity = 7·62 cm. Thickness of walls = 2·54 cm.	4 m.m. Glands, a few enlarged Stroma increased in amount, fairly dense Vessels average	14 m.m.	40	60	About average number, but below average size	Curetted Nov., 1905 Vaginal hysterectomy Feb. 28, 1906	Ch. metritis
	Could not obtain the whole thickness Glands average size and number Stroma dense	19 m.m.	38	62	About average number, but below average size	Vaginal hysterectomy March 21, 1906	Ch. metritis
	3 m.m. Glands average size and number Stroma dense in deep layers, open in superficial	17 m.m.	35	65	About average in number and size		Ch. metritis
Length = 10·16 cm. Length of cavity = 7·92 cm. Thickness of walls = 2·84 cm.	3·5 m.m. Glands increased in number but not enlarged Stroma dense with many cells	21 m.m.	43·1	56·9	About average number, one or two increased in size but not in thickness of walls	Colporrhaphy and curetting Oct., 1905 Vaginal hysterectomy Oct. 31, 1906	Ch. metritis
	3 m.m. Glands average in number, a few enlarged Stroma dense	19 m.m.	34·5	65·5	About average in size and number	Curetting Jan., 1904 Curetting Aug., 1904 Vaginal hysterectomy Nov. 17, 1906	Ch. metritis
Weight = 120·4 gm. Length = 8·25 cm. Length of cavity = 6·9 cm. Thickness of wall = 2·209 cm. Width = 6·09 cm.	2·5 m.m. Glands enlarged and very numerous Stroma small in amount but contains many cells	16 m.m.	40·5	59·5	About average number, a few much enlarged, and have thickened walls owing to fibrous deposit in tunica media	Vaginal hysterectomy Jan. 31, 1906	Ch. metritis
	3 m.m. Glands enlarged and very numerous Stroma loose network	18 m.m.	32·5	67·5	About average in number, below average in size	Curetted 1904 Vaginal hysterectomy April 10, 1906	Ch. metritis
	2 m.m. Glands average size, below average number Stroma loose network	12 m.m.	41·0	58·6	About average number and size	Vaginal hysterectomy May 30, 1906	Ch. metritis
Length = 10·78 cm. Length of cavity 8·89 cm. Breadth = 6·35 cm. Thickness of walls 2·54 cm.	5 m.m. Glands much increased in number and enlarged Stroma small in amount and loose network		38·3	61·7	Average in number and size	Vaginal hysterectomy June 2, 1906	Ch. metritis

Number.	Name. Age.	Social Condition.	No. of Pregs.	Last Pregnancy.	Symptoms commenced.	Menstruation.	Menorrhagia. Metrorrhagia.	Intermenstrual discharge.	Pain.	Clinical Examination.
xxxvii.	E.E. L. 52	M	9	19 yrs. ago	19 years	Since last confinement has had menorrhagia; lately metrorrhagia also	Both	Yellow	None	Uterus anti-flexed, mobile, enlarged, and tender
xxxviii.	E.T. 40	M	5	3 yrs. ago	14 years	Menstruation regular but copious	Menorrhagia	White	Constant aching in both iliac regions worse during menstruation	Uterus enlarged, tender
Uteri with Chronic Metritis										
xxxix.	H. J. G. 40	S	0		16 months	Missed two months, then flooded one month	Both	None	None	Uterus heavy moveable
xl.	Clinical Notes not obtainable									
xli.	Clinical Notes not obtainable									
xlii.	L. C. 27	M	6	3 mths.		Irregular	Both	Yellowish white	Severe in lower part of abdomen during first day of menstruation	Cervix lacerated Uterus large, retroflexed
xliii.	F. G. 28	S	0		2 years	Every 2-3 weeks amount excessive	Both	Constant, copious white	Constant in left side and back for 2 years. Worse during menstruation	Cervix and os conical, stenosed Uterus enlarged, mobile
xliv.	E. N. 42	M	4	16 yrs.	6 months	For 6 months has had irregular hæmorrhage	Both	Dark brown for 6 months	Gnawing pain in hypogastrium for 6 months	Cervix enlarged, pliable Uterus enlarged, mobile
xlv.	E. A. S. 28	M	2	1 year	2 year's ago Much worse since miscarriage 1 year ago	Continuous 4 to 6 six weeks	Both	Yellow, foul smelling	Constant aching in both iliac regions, worse during exertion and menstruation	Cervix large and bossy Uterus enlarged, not adherent

Macroscopical Condition.	Thickness and Description of Endometrium.	Thickness of Meso-metrium.	% Connective Tissue.	% Muscular Tissue.	Blood Vessels.	Operation.	Clinical Diagnosis.
Weight = 106·3 gm. Length 9·52 cm. Length of cavity = 7·62 cm. Thickness of wall = 2·54 cm.	2·5 m.m. Glands somewhat enlarged but few in number Stroma dense, but not very many cells	17 m.m.	50	50	Increased in number, average size	Vaginal hysterectomy Sept. 2, 1906	Ch. metritis
	2 m.m. Glands increased in number & slightly enlarged Stroma dense, contains many dark staining cells There are also numerous large spaces	20 m.m.	41·2	58·8	Increased in number, a few enlarged, but none have thickened walls	Curetted 1899 Curetted 1901 Curetted 1902 Curetted 1904 Curetted June 1906 Vaginal hysterectomy Nov. 9, 1906	Ch. metritis

and some Concurrent Disease.

Length of uterus = 8·63 cm. Length of cavity = 8·02 cm. Weight = 127·5 gm.	1. m.m. A few glands enlarged Stroma dense Vessels few in number	Varies	37·6	62·4	Average number and rather small. Walls not thickened, and contain no fibrous tissue, average amount of connective tissue around groups	Curetted July, 1903 Vaginal hysterectomy July, 1904	Chronic metritis and intramural fibroids
	1. m.m. Glands increased in number and size Stroma dense Veins dilated	13 m.m.	42·1	57·9	About average in number and size	Vaginal hysterectomy	Chronic metritis and carcinoma cervix
Length of uterus = 10·16 cm. Length of cavity = 6·35 cm. Weight = 3·81 cm.		20 m.m.	36·5	63·5	Many vessels enlarged and have thickened walls, due to deposit of fibrous tissue in tunica media	Vaginal hysterectomy March 9, 1905	Chronic metritis and carcinoma cervix
	0·5 m.m.	18 m.m.	48·2	51·8	Increased in number; average size; walls not thickened	Curetted 1899 Double salpingo oöphorectomy May, 1902 Vaginal hysterectomy February, 1903	Chronic metritis and salpingo öophoritis
	0·5—1 m.m. Glands dilated Stroma dense	13 m.m.	48·7	51·3	Average number and size. Walls show no degenerative changes	Curetted November, 1903 Vaginal hysterectomy April, 1903	Endometritis and intramural fibroids
Weight = 127·5 gm. Length = 9·8 cm. Length of cavity = 8·53 cm. Thickness of walls = 1·9 cm.	Whole thickness could not be obtained	20 m.m.	39·1	60·9	Average number and size	Vaginal hysterectomy July 31, 1906	Chronic metritis and carcinoma cervix
	1·5 m.m. Glands about average in number and size Stroma dense, with many large cells	10 m.m.	51·5	48·5		Right ovariotomy 1904 Partial left ovariotomy 1905 Vaginal hysterectomy July 7, 1906	

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Intractable Uterine Hæmorrhage and Arterio-Sclerosis of the Uterine Vessels.*

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Many cases of severe uterine hæmorrhage, occurring near the menopause and without obvious cause, have been reported within recent years. The pathological findings in these cases have been almost as various as the constructions put on the pathological conditions in the attempt to explain the hæmorrhage.

Nearly all have shown thickened blood-vessels, and the vessel changes have been regarded by many observers as the causal factor. In particular, Reinecke, in 1896, reported four cases where hysterectomy was performed for bleeding, and where the pathological examination showed the most marked changes to be in the vessels. He explained the hæmorrhage as due to the inability of the thickened, rigid vessels to respond to vasomotor stimulation. Cholmogoroff, Pichevin and Petit, and more recently Barbour and others have reported similar cases. But the connection between uterine arteriosclerosis on the one hand and uncontrollable uterine hæmorrhage on the other, has not been clearly established. Cases are known where a marked sclerotic condition of the vessels has been unattended by hæmorrhage, and the occurrence of serious hæmorrhage has been noted where the vessels showed no marked change.

Pozzi, in 1899, still seeking the cause of hæmorrhage in the condition of the vessels, differed from other observers in finding the thickening in and around the vessel walls due mainly to an increase in elastic, and only to a less extent, in fibrous tissue. The great increase in the elastic tissue of the organ gave to it, he considered, a peculiar resistance and paralysed its contracting power, so that hæmorrhage readily occurred. Anspach, on the other hand, in a recent paper (1906) demonstrates the physiological increase in elastic tissue in multiparous uteri, and suggests that the cases of bleeding, which, as being in his opinion dependent on a pathological condition of the musculature, he designates 'metrorrhagia myopathica," may be due to a failure in the normal increase of elastic tissue.

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Theilhaber, noting the extreme difficulty of distinguishing physiological from pathological changes in the blood-vessels of an organ subject to such functional variations as the uterus, and believing that the majority, if not all, of the changes described as arterio-sclerosis are conditions normal in a parous uterus, lays stress on the part played by the musculature in the occurrence of hæmorrhage. But exactly the same difficulty in distinguishing what is pathological from what is physiological arises here. What is the normal proportional increase of fibrous tissue to muscular tissue with advancing age and repeated pregnancies? When does Theilhaber's "muscular insufficiency" arise?

Other observers have sought the cause of the hæmorrhages in ovarian changes. That all the causes have a very definite connection with the periodic ovarian stimulation on which menstruation depends is quite clear from the histories. None of the patients manifesting uncontrollable hæmorrhages of obscure origin was past the menopause; and, as Barbour justly remarks, "Arterio-sclerosis after the menopause has been frequently described, but never as accompanied by serious hæmorrhage."

A consideration of the mode of occurrence and control, so far as we know it, of the normal menstrual hæmorrhage, may throw some light on the causation of abnormal uterine hæmorrhages. It is clear that the normal hæmorrhage is brought about and controlled by several factors, and that a disturbance in any one of these may cause irregularities.

That a gradually increasing pelvic congestion occurs and culminates in hæmorrhage mostly by diapedesis but partly also at the height of the congestion by rupture of some of the endometrial capillaries, has been sufficiently demonstrated; as also the fact that this periodic congestion is dependent on the ovaries. Whether the general rise of blood pressure is due to an internal ovarian secretion exerting its influence through the blood, or is determined by nervous influences, or depends on both these factors, is still doubtful. But at least it is safe to assume that variations in the normal stimulation may result in abnormalities of menstruation.

No less important than the ovarian stimulus is the response of the uterus, and here it will be convenient to consider separately the response of the musculature including the blood-vessels, and of the endometrium.

The part played by uterine contractions in controlling hæmorrhage has been fully described by Theilhaber. He points out that during the greater portion of the menstrual period the uterus is large and

flabby; then contractions occur which become gradually longer while the bleeding becomes correspondingly less, until finally the flow ceases completely, strong and continued contraction being necessary for this complete cessation. Similarly in the puerperium the cessation of lochial discharge is dependent on contraction of the muscle. Thus, he argues, if the muscular contraction is insufficient, hyperæmia results with its probable secondary results in swelling and œdema of the uterine parenchyma, and following these, long-continued menorrhagia may readily occur. Anspach explains the actual occurrence of menstrual hæmorrhage as due to obstruction to the venous return, whereby congestion is produced resulting naturally in diapedesis, the obstruction being caused by weak uterine contractions narrowing the veins but failing to narrow in a corresponding degree the thicker-walled arteries. This is essentially the view of Theilhaber, leaving us to infer that the cessation of the hæmorrhage will be brought about by contractions strong enough to close the arteries as well as the veins.

Any explanation of uterine hæmorrhage that even only apparently separates, in regard to its action, the uterine muscle from the blood-vessels, appears to me to be defective. The uterus may be ideally considered as a muscular expansion of the vascular walls, as Keiffer, from his study of the histology of the nervous system in the uterus, points out. Vasomotor stimulation causes contraction or relaxation of the whole uterus. The involuntary muscle of the vessel walls is to be regarded as essentially part of the uterine musculature, subject for the most part to the same variations and the same pathological changes. From this point of view it appears possible that during the height of menstrual congestion the pressure within the vessels is such as to paralyse their contracting power, and that as the pressure lessens, partly from escape of the blood and partly probably from natural diminution of the ovarian stimulus, the vessels regain their tone and the uterus responds to the vasomotor stimulation by contractions sufficient to stop the hæmorrhage. For the complete cessation of the flow strong and lasting contraction is necessary, but this is brought about as a response on the part of the entire musculature to vasomotor stimulation. It follows that any cause interfering with the normal response of the musculature may cause irregularities of menstruation.

The response of the endometrium to the menstrual stimulus is important and interesting. The gradually increasing congestion causes a definite swelling of the mucosa, so that the stroma cells become more distinct in outline and later polyhedral from mutual

pressure, while the glands appear larger from swelling of their epithelial cells. The surface capillaries show a remarkable power of distension to accommodate the increased supply of blood, and appear also to increase in number, so that some observers have thought that new capillaries are formed. But it is possible that the delicate-walled collapsible tubes become apparent only when in a more or less distended condition. The actual hæmorrhage occurs by diapedesis through these delicate capillary walls, and partly also in all probability by rupture of some of them, the corpuscles finally forcing their way between the individual cells of the surface epithelium, or, by dislodging one or two adjacent epithelial cells, finding a freer exit.

What is true of normal hæmorrhage is true also as regards the mode of occurrence of abnormal uterine hæmorrhage. There is no suggestion in any of the recorded cases of severe bleeding that rupture of the presumably diseased vessels had occurred. The hæmorrhage occurs by capillary oozing, an over-distension of the endometrial capillaries being first produced; and the ease with which hæmorrhage occurs depends to a considerable extent on the healthy condition of the capillaries and their resisting power. One would expect, therefore, that any disturbances in the circulation, whether arising from increased arterial supply causing over-filling of the capillaries, or from venous obstruction producing over-distension by backward pressure, would tend to produce uterine hæmorrhage. And there is little doubt that such circulatory disturbances would result in the appearance of hæmorrhage much more commonly than they do were it not for the peculiar character of the endometrium, its unique readiness of response to stimulation.

So far, therefore, the causes of abnormal uterine hæmorrhage fall naturally into three groups:—

1. Abnormalities in the periodic ovarian stimulation.

2. Conditions giving rise to muscular insufficiency, either from
   (*a*) actual deficiency of muscular tissue, or
   (*b*) loss of tone, and consequent deficient response to vaso-motor stimulation.

3. Conditions giving rise primarily to continued congestion of the endometrium, either from
   (*a*) increased arterial supply, or
   (*b*) venous obstruction.

1. Of ovarian changes and their effect on menstruation we know comparatively little. We know from actual experiment that the

growth of the uterus depends directly on the healthy functional activity of the ovaries, that the complete removal of the latter results in atrophy of the uterus and of the genital organs generally, and that the retention of even a small piece of healthy ovarian tissue is sufficient to prevent these regressive changes. Physiologically there is a gradual lessening of ovarian activity as the menopause is approached, and this is accompanied by these general regressive changes, atrophy of the uterine muscle and mucosa, thickening of the walls of the vessels with narrowing of their lumina, and the gradual cessation of menstruation.

Menorrhagia in connection with ovarian disease, especially early cystic changes in the ovary, is well-known to occur, and was noted many years ago by Lawson Tait. The hæmorrhage in these cases is unaffected by curetting or indeed by any treatment save removal of the diseased organ. The endometrium often shows no appreciable change from the normal—a noteworthy point in view of the manifold forms of change which it is liable to show in cases of venous congestion. Exactly how the menorrhagia is brought about is not clear. Brennecke's explanation of the cases which he named "endometritis hyperplastica ovarialis" was based on Pflüger's theory that menstruation is directly dependent on ovulation. But this theory has since been shown to be insufficient, and Brennecke's explanation covered only those cases where lasting congestion and consequent hyperplasia, with menorrhagia as a secondary result, occurred. Czempin recognized cases of severe menorrhagia unconnected with endometrial changes occurring in association with adnexal diseases. These he suggested might be due to an exaggeration of the normal stimulation, resulting in arterial congestion and consequent severe and lasting hæmorrhage. Since the menstrual stimulus is associated essentially with an increased blood pressure, producing its effects in the uterus by creating a sudden hyperæmia, it seems reasonable to suppose that an exaggerated stimulus should result in menorrhagia.

The probability of disturbed ovarian function occurring at the menopause is readily appreciated, and the marked connection between the approaching menopause and uncontrollable uterine hæmorrhage strongly suggests the possibility of ovarian changes being a causal factor in the hæmorrhages. Cases of spontaneous cure occurring when ovarian activity ceases and the menopause is fully established, strengthen this possibility. Gardner and Goodall, in a recently published paper, refer to such cases, and point out that if ovarian stimulation acting on a uterus affected by "chronic metritis"

is the cause of the menorrhagia, then the menorrhagia should continue until ovarian function ceases, the condition of the uterus remaining unaltered. But they report a case where severe menorrhagia occurring near the menopause and associated with all the usual signs of "chronic metritis" disappeared under conservative treatment, yet normal menstruation indicating ovarian activity, remained.

It is interesting to note that several of the reported cases of uncontrollable hæmorrhage laid to the charge of arterio-sclerosis or muscular deficiency have shown ovarian changes. Thus, in the case reported by Barbour, the right ovary was cystic with very little ovarian substance left, while the left was small and sclerosed; and of Anspach's three cases of "metrorrhagia myopathica" two showed ovarian changes to a slight extent.

Further, it is evident that impairment of the functional activity of the ovaries may occur independently of any demonstrable lesion, and may be the cause of hæmorrhage in those cases where no pathological condition is found. Indeed, Freund goes so far as to say that only in cases of bleeding from carcinoma has an anatomical basis for the hæmorrhage been demonstrated.

2. It is obvious, from a consideration of the part played by the musculature in the control of menstrual hæmorrhage, that failure of the muscle to contract efficiently may be associated with hæmorrhage of the severest kind. Such failure may be due to

(*a*) actual deficiency of muscular tissue. Thus, insufficient muscular development, such as not infrequently obtains at the time of puberty; or actual atrophy and degeneration, such as occurs normally at the menopause but may occur as a premature and so far pathological change; or "sclerosis" of the uterus, whether due to a primary diathetic condition (Brionde, Richelot) or a secondary result of infection (Bland-Sutton), all come under this heading, and in all the muscular coat of the vessels participates in the pathological changes. Without actual deficiency of muscle, insufficient contraction may be due to

(*b*) loss of tone in the musculature. This may occur as a simple atony associated with a lowering of tone throughout the body from some general cause, and possibly then accompanied by manifestations of muscular atony in other parts of the body, gastric symptoms, etc.; or as a local change from the various causes producing atony of the uterine muscle either directly or through fatigue of the nerve-cells. The effects of various poisons, of alcoholism, etc., on the nerve-cells has not yet been shown, but these have possibly a direct effect in producing atony of the musculature.

In cases of menorrhagia arising from this cause, intermenstrual leucorrhœa is a not infrequent symptom, the explanation being the lowering of tone in the vessel walls and the consequent ready escape of leucocytes. It is doubtful whether muscular insufficiency alone, apart from this element of lowering of tone, ever gives rise to leucorrhœa, since the capillaries in a healthy condition have a remarkable power of resisting pressure, and accommodating themselves by enormous dilatation to an increased mass of blood. Typically, deficiency of the musculature, without deficient vitality of the vessel walls from loss of tone, will result in menorrhagia, the hæmorrhage becoming more prolonged and profuse as the secondary hyperplasia of the uterine parenchyma becomes more evident.

It is in this class that Theilhaber would place the great majority of the cases of uncontrollable hæmorrhage occurring at the menopause, and in this he is enthusiastically supported by Palmer Findley. Normally, the regressive changes in the musculature associated with the menopause are accompanied by corresponding regressive changes in the blood-vessels which become accommodated to a decreased blood-supply. Anything, therefore, preventing this normal diminution in blood supply, anything keeping the blood supply at its former level in face of the retrograde changes in the muscle, will cause hæmorrhage—hæmorrhage which these authors regard therefore as due to muscular insufficiency. But the regressive changes in the musculature are admittedly physiological, normal at the menopause, and would not give rise to hæmorrhage were it not that the vessels are prevented from closing proportionately. It would seem more logical, then, to regard the hæmorrhage as a result, not of the atrophied condition of the muscle which is physiological, but of the patent condition of the vessels which is pathological. It is evident that a normal blood supply acting on an insufficient musculature will have the same practical result as an increased blood supply on normal musculature. The question is whether the muscular atrophy is premature and therefore pathological, and justly to be blamed as the causal factor in the hæmorrhage, or whether the blood supply is being maintained at a pathologically high level, either through failure to diminish when it naturally should, or from a real and as yet unexplained, increase in pressure.

Although many of the reported cases of intractable hæmorrhage show intermuscular fibrosis, with atrophy and degeneration of muscle, yet there is no general agreement that atrophy of muscle, beyond that occurring normally at the menopause, is characteristic. The point is extremely difficult to determine. Thus, in four cases re-

ported recently by Wittek three show intermuscular fibrosis, and the fourth does not. But the exceptional case was a nullipara. Shaw, investigating cases of chronic metritis, finds no marked increase of connective tissue at the expense of muscular, but rather a hypertrophy of both elements due, he considers, to efforts on the part of the uterus to rid itself of the thickened mucosa.

Cases of hæmorrhage where the musculature is insufficiently developed, or where its atrophy is definitely premature, are undoubtedly to be placed in the category of myopathic hæmorrhages. In this class also may justly be placed those cases which have as their starting-point an acute febrile disease such as typhoid; or are associated with anæmic conditions, particularly chlorosis, or with chronic wasting diseases, such as phthisis. All these are definitely associated with changes in involuntary muscle, and show their effects as a rule on the heart. Otherwise, changes in the uterine musculature are probably for the most part secondary to circulatory disturbances.

3. Cases of intractable uterine hæmorrhage due primarily to circulatory disturbances form an important group. Continued congestion of the endometrium may be due to

(*a*) an increased arterial supply. In determining this, the endometrium may be primarily at fault, as in cases of direct infection, placental retention where the remnants act the part of an irritant foreign body, and malignant disease of the mucosa. Inflammation and tumours of the adnexa, as also tumour formation, particularly fibromyoma, in the uterus itself, will likewise determine an increased arterial supply, and to these may be added any local irritation, mechanical or psychical. Histologically, as Freund has shown, this arterial congestion will be characterized by an increase in the number of capillaries, and a widening of the vessels, in which naturally the veins take part more readily and to a greater extent than the arteries, the widening in the veins being of a diffuse character affecting mostly the capillary venous network on which the strain of the increased blood supply first tells.

(*b*) Venous obstruction may arise from a general condition, such as heart or kidney disease, chronic lung affections, chronic constipation, etc.; or from local obstructions to the venous return, such as would be caused by tumours within or without the uterus; or from displacements of the uterus. The resulting passive congestion does not, according to Freund, cause any increase in the number of capillaries, but results first in irregular dilatation of the larger veins, particularly

of those in the fundus; the pressure may further tell back on the arteries and produce thickening of their walls.

Long-continued congestion, from whatever cause arising, brings about a very typical reaction in the mucosa. There appears to be little doubt that a large number of the cases variously described as "hyperplastic endometritis," "hypertrophic glandular endometritis," "interstitial endometritis," etc., are simply, as Van Meerdervoort describes them, secondary results of long-continued congestion of the mucosa. The differences in the various forms, including "fungous" and "polypoid endometritis" are merely of degree, and he suggests "chronic œdema" as a more correct and appropriate title for the whole class. Löfquist arrives at very similar conclusions, suggesting that all the various changes are degrees of what he terms "decidual reaction." The changes are slow and gradual; the distension of the capillaries does not result in hæmorrhage, as in the more rapid menstrual increase in blood pressure, because the tissues have time to adapt themselves to the increased blood supply. Hyperplasia of the tissues results, affecting first the mucous membrane and extending in the course of time to the entire uterus, giving one of the forms of "chronic metritis."

Many attempts have been made to elucidate "chronic metritis," some observers regarding it as due always to infection of a more or less remote date, others as secondary to chronic endometritis, others as a primary condition giving rise to chronic endometritis by interference with muscular contraction and consequent hyperæmia. That there are many forms of pathological change involved and so far confounded is probable. But one form of the affection—where the uterus is generally enlarged and soft, with thickened walls showing no evident disproportional increase in fibrous tissue, but rather a general swelling and hyperplasia of all the constituent elements, the endometrium at the same time exhibiting one or other of the usual forms of "glandular endometritis"—is almost certainly due primarily to a disturbance in the circulation. This is the form of chronic metritis found in the various displacements of the uterus.

Clinically, these cases are characterized by a gradually increasing menorrhagia, and this is what we should expect from the pathological condition. The menstrual stimulus, normally resulting in a comparatively rapid over-distension of the capillaries, produces its effect more slowly through the thickened mucosa with its already comparatively distended and more numerous capillaries, but when hæmorrhage does occur, at the very height of the congestion, it is more profuse than normal because of the more widely distended

condition of the capillaries. Similarly the bleeding will last longer, partly from the lowered tone of the vessels from prolonged dilatation, and partly from inefficiency of the œdematous muscle, together resulting in less efficient response to the vasomotor stimulation, and so failure to arrest the hæmorrhage with normal rapidity.

But not all cases of venous congestion become clinically apparent as menorrhagia. Whether abnormal hæmorrhage results appears to depend to some extent on the resisting power of the endometrial capillaries. The vessels of the endometrium are peculiar. Macgregor has described them carefully, distinguishing between the "thick-walled" and the "thin-walled" vessels. The latter are merely tubes lined by a single layer of delicate endothelium, and show enormous power of dilatation. The "thick-walled" vessels, on the other hand, derive support from a condensation of the stroma cells around them, and these, Macgregor states, while showing many changes in the way of thickening of their walls, hyaline degeneration, etc., never dilate. This is open to question. The thick-walled vessels, which it seems hardly correct to call "arterioles" may become dilated in cases of long-continued venous congestion. This was well shown in a recently observed case of retroflexion in which there had been no abnormal hæmorrhage. The mucous membrane was very markedly thickened, giving rise on curetting to the suspicion of malignancy, and its appearances were those of an advanced "chronic œdema," with great increase in the number of glands and swollen stroma cells showing their outlines and anastomosing processes in great perfection. Most striking was the large number of dilated "thick-walled" vessels, of globular outline and with a marked condensation of the stroma around them. The condition was produced in all probability by backward pressure from the long over-distended, thin-walled capillaries, the healthy nervous tone of the capillary vessels preventing the relief of pressure by diapedesis. The patient in this case was comparatively young, 32, and unmarried. Where the congestion arises from an increased arterial supply, there is a tendency to metrorrhagia in addition to the menorrhagia commonly observed. Any sudden stimulation, mechanical or otherwise, determining a sudden further increase in the blood supply, may cause hæmorrhage. We may suppose that the explanation is a sudden dilatation from vasomotor paralysis of the vessels formerly in a state of exaggerated tonus.

Influence of the menopause. In determining the causal factor in any case of intractable uterine hæmorrhage, therefore, many conditions must be considered. After excluding the cases probably

arising from muscular insufficiency and eliminating all the usual causes of long-continued congestion, there still remains a class unexplained. Yet a careful consideration of the possible known causes markedly reduces the number of these obscure cases. Thus the patient whose case is reported by Pichevin and Petit, had had typhoid at 30, followed by menorrhagia, which was, however, cured by a supervening pregnancy, but returned in a severe form demanding hysterectomy after another pregnancy two years later. Again, of Pozzi's cases, while the elastic tissue proliferation on which he lays stress may mean nothing more, according to recent researches, than that the uteri examined were parous, one case had had typhoid, and the other had had chlorosis, and at the time of operation had albuminuria, an important symptom of increased blood pressure. It is of further interest to note that none of these patients was very near the menopause.

Those cases becoming evident only at the menopause have probably their origin in circulatory troubles, the congestion having lasted perhaps for years previously without giving rise to any troublesome symptoms.

We do not know how ovarian activity acts in maintaining the uterus in a healthy condition, but a twofold action at least suggests itself. The periodic vasomotor stimulation causing reflex contraction maintains the musculature in an efficient working condition; and ovarian secretion in some obscure way preserves a healthy uterine tone. When ovarian stimulation begins to fail, the uterus suffers in this twofold way, so that there is a certain amount of atrophy from disuse, and a tendency to degeneration from the cutting off of some trophic influence. These effects will be equally produced in a uterus already affected by hyperplasia from continued congestion, and they are sufficient to determine the disturbance of compensation which results in menorrhagia. The menstrual stimulation is essential in starting the hæmorrhage; the prevailing uterine conditions prevent its normal arrest.

Pathological changes in the uterine blood vessels, and their influence in determining hæmorrhage. So far the vessels have been dealt with as essentially part of the uterine musculature, and in no way to be regarded as of primary importance in the causation of hæmorrhage. But arterio-sclerosis has been so extensively regarded as the causal factor that the subject deserves special attention.

There can be no doubt that the vessel changes designated "arterio-sclerosis" are in many cases normal changes, and in no way to be regarded as responsible for the occurrence of uterine hæmorrhage.

Exactly similar changes are found unconnected with hæmorrhage. Marked degenerative changes, designated by Anspach "periarterial" and "perivenous" degeneration occur normally in every parous uterus. In a specimen stained by hæmatoxylin and eosin these areas appear pink with few and scattered nuclei; with Van Gieson's picro-fuchsin method they appear a bright yellow, in clear contrast to the yellow of surrounding muscle; and with Weigert's resorcin-fuchsin stain for elastic fibres they take a dark blue or black colour. This definite increase in elastic tissue round the vessels is characteristic, and appears to occur during the puerperium. The elastic tissue of the uterus generally is increased with every pregnancy, so that it is always possible to tell a parous from a non-parous uterus in a Weigert preparation. The fine elastic fibrils become thickened and curled, and show a tendency to clumping. In the vessels, the inner elastic lamina no longer appears as a clear unbroken wavy fibre, but is thickened and broken up, and there is an increase of elastic fibrils throughout the media. Melnikow-Raswendenkow believes that the place of lost parenchyma in any organ is taken by elastic tissue. Pick records a case of uterine hæmorrhage resisting treatment in a woman of 63, which may have been due to an evident failure in the normal increase of elastic tissue around the uterine vessels.

The normal involution of arteries after pregnancy is to be distinguished from a pathological change. Naturally there is enormous hypertrophy of the entire musculature in the gravid uterus, with new formation of blood-vessels, many of which must undergo entire obliteration during the process of involution. This is brought about by a process recognised as "endarteritis obliterans," in which there may be enormous overgrowth of the intima, practically occluding the vessel; contraction of the new-formed connective tissue throughout the media and in the adventitia helps the process. Normally there is a slight increase of fibrous tissue at the expense of muscle when involution is completed; the vessels share in this, their adventitial coats being relatively slightly thickened after each pregnancy. It appears, therefore, that thickening of the intima, with irregular increase in its elastic tissue, thickening of the media, with increase in the fibrous tissue of both media and adventitia; later, degeneration of the coats and of the surrounding tissue with deposition of elastic tissue, may all occur as physiological changes.

The following case, in which the uterus was removed on account of severe menstrual pain, and which is probably primarily one of glandular endometritis due to infection, possibly syphilitic, the changes in the musculature being secondary, shows vascular changes

which may be taken as physiological for a uterus with a corresponding history:—

Mrs. R., aged 36, was admitted to the Dundee Royal Infirmary on December 4th, 1905. She had been married 18 years, and has had 9 pregnancies, the last 5 years ago. The first 2 pregnancies resulted in miscarriage at the 3rd month, the 3rd was normal, the 4th was premature (twins at the 6th month), the 5th ended in a miscarriage at the 5th month, the 3 following were normal, and the last ended in a miscarriage at the 5th month. Menses began at 14, occurring regularly every 3 to 4 weeks, unaccompanied by pain. She had some trouble which she described as a "growth of the womb" after the 7th pregnancy 8 years ago, but this disappeared under treatment by "douches and medicine." Since the last pregnancy she has not felt well, and for more than a year has had severe menstrual pain which has become much worse during the last two or three months. The pain lasts from two days to a week, beginning before the onset of the discharge; is worst for 2 or 3 hours after the onset, and continues after the flow ceases. The pain makes her sick. The discharge is normal in amount and character, and there is no intermenstrual discharge. She suffers from severe headaches and occasional frequency of micturition.

On admission, she was found to be thin and pale, and as the physical examination gave rise to the suspicion of interstitial fibromyoma of the uterus, hysterectomy was decided on.

Operation. Professor Kynoch removed the enlarged uterus by the vaginal route. The patient made an uninterrupted recovery.

Path. No. 4559. The specimen consists of the uterus, which is generally enlarged, measuring after hardening in alcohol $10\frac{1}{2}$ cm. × 5 cm. × 5 cm. On section, the uterine walls are seen to be thickened, and the endometrium to be strikingly thickened. The uterine cavity is 8 cm. long; the anterior wall at its thickest part measures $2\frac{1}{4}$ cm., and the posterior $2\frac{1}{2}$ cm. The mucous membrane shows a smooth undulating surface of a pinkish colour, and is of normal thickness in the cervix, greatly thickened just above the internal os, and less markedly thickened towards the fundus; at its thickest part it measures on the anterior wall 7–8 mm., and on the posterior 9–10 mm.

Histological examination: mucous membrane. The most marked change is the great increase in the number of the glands, a few of which are dilated. The glandular epithelium is swollen and œdematous. The stroma is loose and œdematous, and the vessels show marked condensation of the stroma around them. The junction

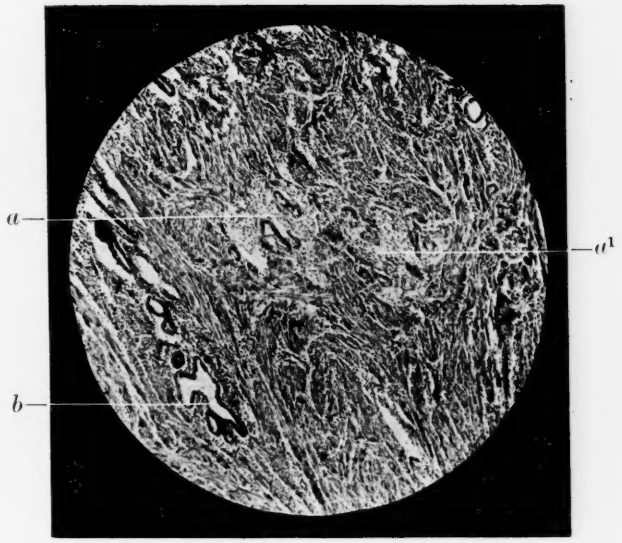

Fig. 1. Path. No. 4559. No hæmorrhage. "Periarterial" degeneration in small vessels near the mucosa. Note also the unusually deep penetration of the glands into the muscle, and the cellular infiltration between muscle bundles. Hæmatoxylin and eosin. (×50.)

a, *a*1, Degenerate areas round small arteries. *b*, Glands penetrating musculature.

between mucosa and muscle wall is strikingly irregular, glands penetrating the muscle to an unusual depth, and carrying with them strands of stroma (Fig. 1).

Musculature. The proportions of muscular and fibrous tissue appear about equal. The tissues are œdematous the muscle nuclei appearing swollen, and patches of embryonic cells are seen throughout the musculature between the muscle bundles.

Vessels. The larger arteries are thickened in all their walls. The lumen is irregular in many cases, from projection of the media and occasionally of the intima. With Weigert's stain these irregular projections of the intima are seen to contain a good deal of elastic tissue, and the thickened inner elastic lamina is well shown, as also the deposition of elastic tissue in and around the degenerated adventitia. The intimal thickenings are shown to be almost entirely fibrous by Van Gieson's stain, and the degenerated areas around the vessels appear bright yellow. The media appears rather poor in nuclei, and the degenerated areas are almost free of nuclei, in a hæmatoxylin preparation. The " periarterial " degeneration is very marked in some parts, especially in the smaller vessels near the endometrium (Fig. 1). Some of the arteries show a remarkable proliferation of the intima causing almost complete obliteration of the lumen. The veins show similar changes. There is an irregular proliferation of the inner coat, with an increase in fibrous tissue, and a thickening of the outer coat with degeneration extending into the surrounding tissues—" perivenous " degeneration (Figs. 6 and 7).

The case is of interest in several ways. The enormous hypertrophy of the endometrium in the absence of any tendency to hæmorrhage, and the marked penetration of the glands into the underlying muscle without in the least suggesting a malignant process, are evidences that the primary trouble is in the endometrium. Cornil noted this penetration of the glandular elements as characteristic of chronic metritis. The cause of the pain is obscure. Possibly it was simply an exaggeration of the pain commonly experienced at the height of menstrual congestion : possibly the much thickened mucosa, acting as a foreign body, induced more severe contractions than normal in the endeavour of the uterus to expel the foreign body, and the increased blood pressure from ovarian stimulation in the premenstrual stage started the contractions which became more severe as the congestion increased. The increase in the musculature may then be of the nature of a true " work-hypertrophy." The existence of patches of embryonic cells throughout the entire muscle wall, but

most commonly in its inner half, is noticeable, and suggests extension of an infective process from the endometrium. Probably the case is to be classified as one of chronic metritis secondary to chronic infective endometritis, the musculature being as yet but slightly affected by the infective process.

The senile changes occurring in the uterine vessels are illustrated in the following case:—

Mrs. D., aged 51, millworker, was admitted to the Dundee Royal Infirmary on 7th May, 1906. She has had 7 pregnancies, all normal, the last 21 years ago. Menses began at 13, and occurred regularly every 28 days, lasting for 2 days, the discharge being scanty and accompanied by slight pain; the menopause occurred 4 years ago. For 2 years there has been a white discharge, fairly copious, and lately the discharge has been bloody at times. She had been in the Infirmary in the beginning of 1905 with symptoms of mitral incompetence, some chest trouble and diarrhœa suggesting enteritis, and again in February 1906, with gastro-intestinal symptoms.

Physical examination. Patient is a worn-out old woman, and was intoxicated on admission. She has a slight cough; there are dulness and tubular breathing without accompaniments at the left apex. There is a soft systolic mitral murmur. She complains of frequency of micturition. Examination under chloroform showed the cervix to be much shortened, the vaginal aspect normal except for small submucous hæmorrhages. There was bleeding from the orifice. A swelling, possibly the fundus, was felt lying to the left; and, suspecting malignant disease of the body of the uterus, Dr. Buist decided to do vaginal hysterectomy.

Operation. Dr. Buist operated on May 18th. An incision was made in the posterior fornix and into the pouch of Douglas. A tumour mass was found on the left side, nodular, and not very mobile. The right broad ligament was crushed and divided, and the fundus brought down, when the uterus was seen to be small and senile, but otherwise apparently normal.

The tumour mass could not be brought down, and median cœliotomy showed it to be in the sigmoid flexure of the colon with the left uterine appendages adherent. The adhesions were crushed and divided and the hysterectomy completed. The sigmoid tumour was isolated by Renton's clamps and removed, and the bowel sutured end to end.

The condition of the patient after the operation was never very hopeful owing to the chest condition, and on the 21st, an offensive

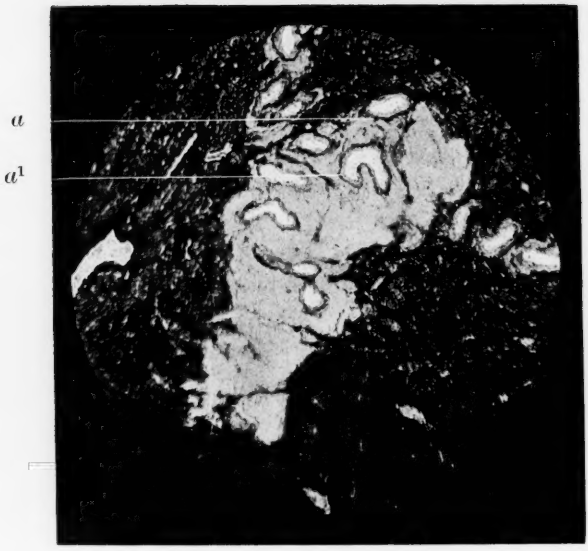

Fig. 2. Path. No. 4750. Senile case. Extensive "intramural" and "periarterial" degeneration in vessels near the mucosa, with marked narrowing of lumina. Van Gieson. The degenerated areas stand out in sharp contrast to the surrounding tissue which is extensively fibrous. (× 65.)

a, a^1, Narrow ring of fibrous tissue, remains of adventitia.

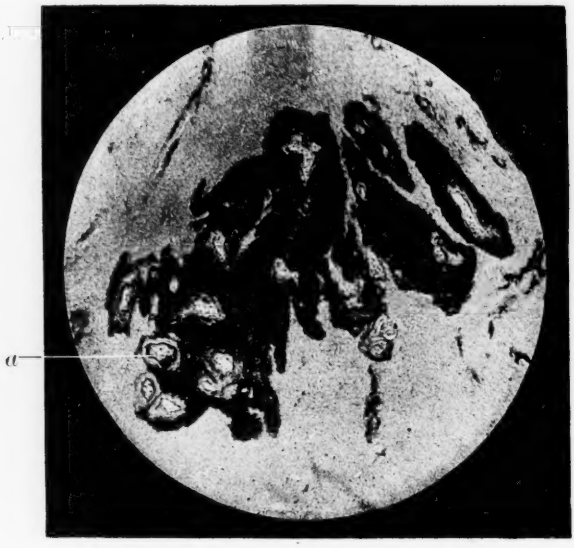

Fig. 3. Same group of vessels as shown in previous figure. Weigert's elastic stain. Note persistence of internal elastic lamina, and presence of elastic tissue, which appears black, in area corresponding to light (yellow) area in previous figure. (× 65.)

a, Inner elastic lamina.

vaginal discharge set in, becoming later putrid and black in colour, and death occurred on the 22nd.

Path. No. 4750. The specimen consists of the uterus which is normal in contour but smaller than usual, measuring 6 cm. × 3¼ cm. × 2¾ cm. The cervix appears normal save for a few subepithelial hæmorrhages. The vessels entering the uterus are visibly thickened and prominent, and on section of the uterus the vessels stand out very distinctly in the vascular zone at the junction of the middle and outer thirds of the wall. Within this zone, extending into the mucosa, the muscular wall presents a curious, firm, homogeneous appearance. The mucosa is atrophied, and stained with blood in some places; its limits cannot be distinguished by the naked eye. The uterine wall measures 1½ cm. in thickness, and is firm.

Histological examination: mucous membrane. This is atrophied to a considerable extent, the glands persisting in groups between which fibrous tissue strands run right up almost to the surface epithelium. In many parts fibrous strands run along under the epithelium. The persisting glands are small, with swollen epithelial cells. There is considerable extravasation of blood into the mucosa, while here and there red blood corpuscles may be seen making their way to the surface between the individual cells of the intact surface epithelium. The stroma is dense and its component cells are swollen and œdematous; fibrous strands invade it in all directions.

Muscular wall. The muscle is everywhere separated up by bands of fibrous tissue. The muscle shows evidence of degeneration and atrophy, its nuclei, especially near the endometrial surface, being swollen and degenerate-looking, and occasionally fragmented.

Vessels. The vessels occur in characteristic groups, with very narrow lumina (some appear entirely obliterated) and a wide area of degeneration surrounding them (Fig. 2). These areas are so extensive that neighbouring areas closely approach each other, and give a very striking appearance to the stained section, in which the degenerated tracts are readily recognized by the naked eye. The process is most marked in the middle third of the muscular wall (the homogeneous area). These areas stain darkly with Weigert's stain, although no definite structure can be made out in them (Fig. 3), and they appear bright yellow, in contrast to the yellow of the muscular tissue, in Van Gieson preparations, with an occasional remnant of red, fibrous tissue, persisting in the midst of the structureless yellow mass (Fig. 2). The larger vessels of the vascular zone show various degrees of thickening and degeneration of their walls. Many show calcification commencing between the intima and the media (Fig. 8).

The intima is not particularly affected throughout; with Weigert's stain it shows a thickening and breaking up of the internal elastic lamina. There are irregular projections into the lumen in many cases, but these appear to be due mostly to an irregular proliferation of the media, over which the uniformly thickened intima extends. Both media and intima are degenerated, nuclei being very few and far apart. The adventitia is thickened throughout, and the degenerated areas in and around the adventitia stain deeply with Weigert's elastic stain. The increase in the adventitia appears to be due chiefly to elastic tissue, with which the media also is spun through, the elastic fibres again blending into a definite layer at the internal elastic lamina. The veins show similar changes. There is an increase of fibrous tissue in the inner coat, and a marked deposition of elastic tissue in the degenerated outer coat. There was thrombosis of a branch of one of the ovarian arteries where it entered the uterus at the broad ligament attachment—a thrombosis evidently of old standing since there was organisation with formation of new vessels in the midst of the thrombus.

The senile change in this case was very advanced considering the age of the patient, but the fact that when admitted to Hospital she was in a state of intoxication, throws some light on the cause of the premature senility. From the histological examination, the case appears to have some points in common with those cases reported as "apoplexia uteri" by von Kahlden and others, where there was hæmorrhagic infiltration of the endometrium associated with sclerosis of the uterine arteries.

The occurrence of the leucorrhœa, beginning two years after the menopause, is undoubtedly to be associated with the disturbed compensation in the heart, which became evident about the same time. Later, the venous obstruction resulting from the sigmoid cancer added to the circulatory disturbance in the uterus, and the discharge thereupon became hæmorrhagic and more troublesome and persistent.

It is evident, therefore, that even very advanced premature senile change in the uterine vessels, with calcification in some of the arteries and consequent rigidity, degeneration of vessels throughout the entire uterine wall, along with marked fibrosis of both musculature and endometrium, are all insufficient of themselves to cause serious hæmorrhage, even in the presence of an incompetent heart. It is equally evident that in a normal senile uterus, an incompetent heart and venous obstruction such as would arise from cancer of the sigmoid flexure would not necessarily give rise to any uterine symptoms. The

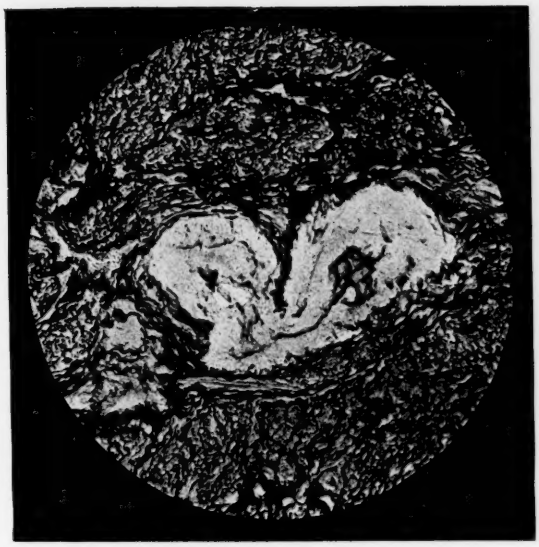

Fig. 4. Path. No. 2505. Menorrhagia. Extensive "intramural" and "Periarterial" degeneration in vessel from vascular zone. Van Gieson. Lumen almost obliterated. Note persistence of fibrous shreds (black) in midst of light degenerated area. (× 130.)

Fig. 5. Same artery as Fig. 4. Weigert. Elastic tissue (black) appears in place of the light area, and at one side this mass appears almost fused with the internal elastic lamina. (× 130.)

A hæmatoxylin and eosin preparation of the same artery shows an appearance closely resembling that in Fig. 1—a light area round the vessel, with few nuclei.

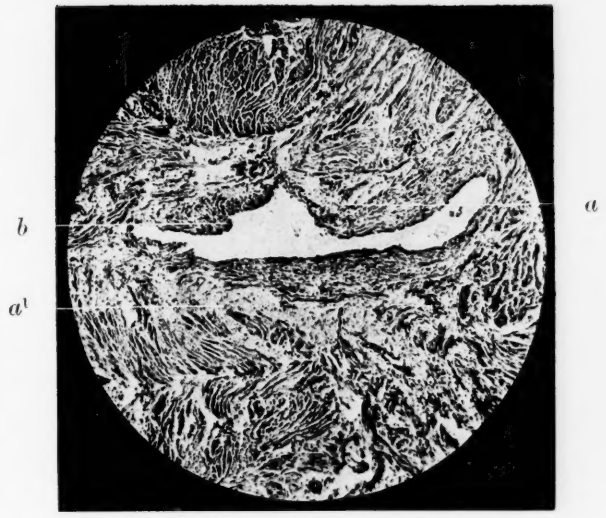

Fig. 6. Path. No. 4559. "Perivenous" degeneration in large vein. Note slightly irregular proliferation of the inner coat. Degenerated area has the same appearance as in "periarterial" degeneration. Hæmatoxylin and eosin. (× 50.)

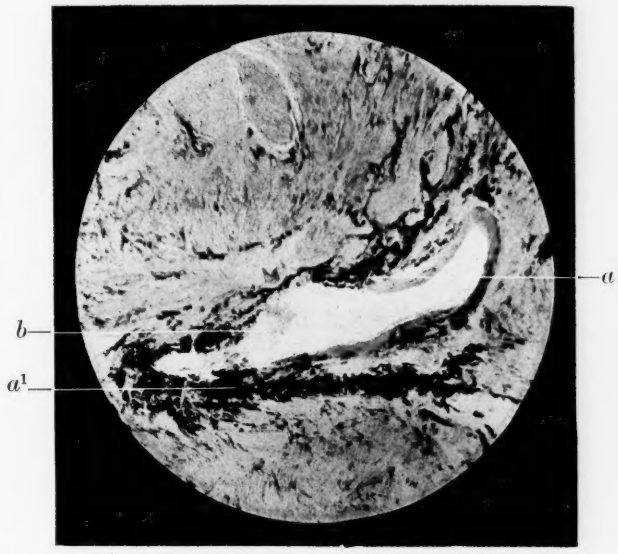

Fig. 7. Same vein as in Fig. 6. Weigert. Note elastic tissue in outer coat, and fine fibrils throughout the irregularly thickened inner coat, as also in all places appearing degenerated in previous figure. A section stained by Van Gieson, of same vein, shows the corresponding areas bright yellow. (× 50.)

a, *a*¹, Degenerated areas in and around outer coat. *b*, Irregularly thickened inner coat.

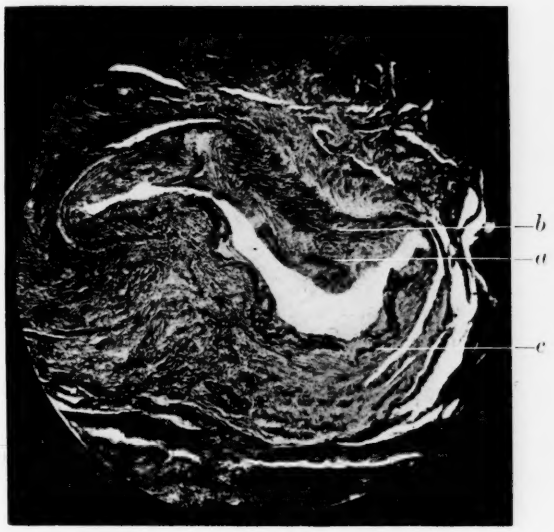

Fig. 8. Path. No. 4750. Senile case. Calcareous degeneration beginning in superficial layers of media in large artery. (×30.)

a, Intima. *b*, Media. *c*, Calcifying area.

local condition determining the discharge in this case is probably the premature degeneration of the vessels, diapedesis through their degenerated and "toneless" walls, in presence of a sluggish arterial supply on the one hand and venous obstruction on the other, being unusually easy. Alcoholism as a factor in bringing about this premature decay of the vessels is well known, but how the effect is produced is still obscure. The absence of the periodic menstrual stimulation in this case sufficiently accounts for the absence of sudden severe hæmorrhages.

Several cases have been reported where severe uterine hæmorrhage was associated with general arterio-sclerosis. But this association does not simplify matters at all except so far as to make it probable that the uterine arterio-sclerosis found was of a similar nature to the general change. Many cases of general arterio-sclerosis never show uterine hæmorrhage. So in cases of bleeding where there is a history of syphilis many years previously. Syphilis we know to be one of the most common causes of general arterio-sclerosis. But by no means all cases of syphilis are attended by uterine hæmorrhage. What determines the occurrence of the hæmorrhage in some cases?

The following case is of interest:—

E.M., aged 50, unmarried, was admitted to the Dundee Royal Infirmary on April 30th, 1906. One pregnancy (twins, 6 months) 20 years ago, since which time she has been blind (coloboma, chronic iritis, cataract). Menses began at 15, and occurred every 3 to 4 weeks, lasting 7 days. Irregular bleeding began in November 1903, the first hæmorrhage lasting 7 weeks, unaccompanied by pain, the second coming on after an interval of 5 weeks and lasting for 4, and the third after an interval of 6 weeks lasting for 5 weeks, at which time she was curetted, the curettings showing nothing noteworthy. The physical examination at this time showed the os to be patulous, edges œdematous and slightly rough; the fundus was nearly 3 inches above the symphysis; there was slight tenderness in the left iliac region, and here the left tube could be palpated as a smooth, fairly firm, elastic oval swelling. There was uterine hæmorrhage on manipulation. The bleeding recommenced 11 days after the curettage, and recurred at intervals up to the time of admission, the last hæmorrhage having begun 2 months before admission and continued up to that time.

Physical examination. Patient is very weak, giddy, and faint. There is slight pain all over the abdomen. She is pale, with dilated capillaries on the cheeks. Pulse 104, of "water-hammer" variety;

and examination of the heart shows presence of aortic systolic and diastolic murmurs.

Operation. Hysterectomy was performed by Dr. Buist a week after admission. A perivaginal incision was made, and a roughly "banana-shaped" hydrosalpinx of the left side removed, clear fluid escaping. The left ovary, which was slightly cystic, was crushed and removed; and the normal right ovary was left. The patient died on the second evening after the operation, and the post mortem examination showed widening of the aorta, with some atheroma; the kidneys contracted and unequal in size, and the liver fatty.

Path. No. 4738. The specimen consists of the uterus, and the cystic left tube. When inflated the tube appears roughly to be equal in size and similar in shape to a banana, with an extremely thin, smooth, transparent wall.

The uterus is of normal contour but generally enlarged; the serous surface is smooth and free from adhesions. The lips of the cervix are somewhat hyperæmic and very slightly nodular; the cervix is $3\frac{1}{2}$ cm. broad, and shows a transversely oval aperture measuring $1\frac{1}{2}$ cm. × 1 cm. The uterus measures $9\frac{1}{2}$ cm. × $5\frac{1}{2}$ cm. × $4\frac{1}{2}$ cm.; the cavity from fundus to external os measures $7\frac{1}{4}$ cm.; in breadth, from the centre of the canal outwards in the direction of the right cornu, it measures $1\frac{1}{2}$ cm., but in the direction of the left cornu, owing to the hydrosalpinx, is reduced to $\frac{3}{4}$ cm. The muscle appears normal, though very pale, and the blood-vessels appear distinctly on the cut surface. The mucous membrane is uniformly smooth and pale, measuring 1–2 mm. in thickness on the anterior wall which itself measures $2\frac{1}{2}$ cm.

Histological examination: mucous membrane. The surface epithelium is intact and smooth throughout the greater part of the cavity; towards the fundus it shows a tendency to become polypoid. The glands are not increased in number, but appear larger than usual, with very markedly swollen epithelial cells. The stroma is œdematous and its cells swollen, and many large dilated blood spaces lined by a single layer of endothelium are to be seen just beneath the surface epithelium. The "thick-walled" capillaries appear normal in many cases, but some are thickened by condensation of the stroma around them, and one or two show hyaline degeneration (Fig. 13). Vessels having definite fibrous and muscular walls are found within the mucosa, occurring often quite close to the glands (Fig. 12), and in sections stained by Weigert's stain for elastic fibres, these show a well-marked inner elastic lamina.

The junction between mucosa and muscle is sharply marked, and

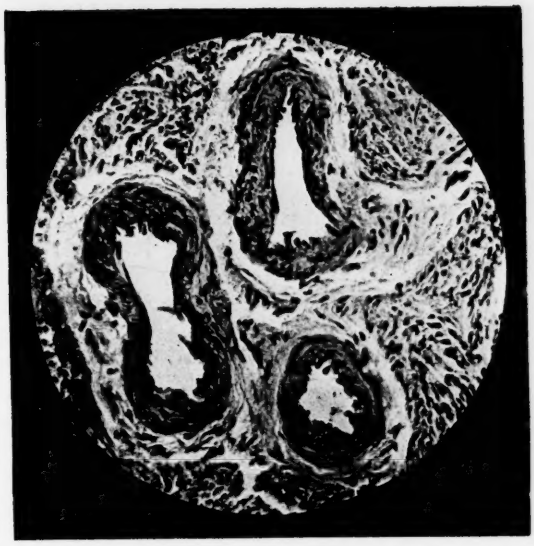

Fig. 9. Path. No. 4738. Arterio-sclerotic case. Severe hæmorrhage. Group of medium-sized arteries showing thickening of all their coats, the adventitia being composed of a lightly staining, sparsely nucleated fibrous tissue. This is clearly a different condition from the "periarterial" degeneration seen in previous figures. Hæmatoxylin and eosin. (× 230.)

Fig. 10. Similar group to that shown in Fig. 9. Van Gieson. The fibrous tissue of adventitial coats and surrounding the vessels appears black. Note that this fibrous tissue does not invade the surrounding muscle to any extent. Note also the fibrous strands throughout the media, and the marked irregular thickening of the intima. (× 60.)

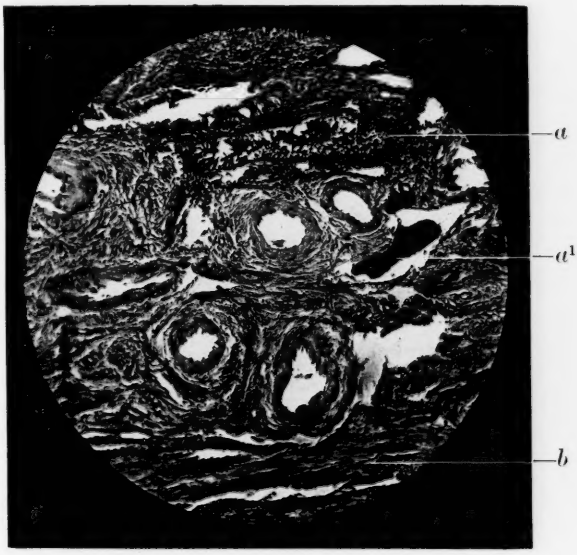

Fig. 11. Same case as Figs. 9 and 10. Group of thickened vessels occurring just under the mucous membrane. (× 90.)

a, Mucosa. a^1, Detached glandular epithelium. *b*, Muscle.

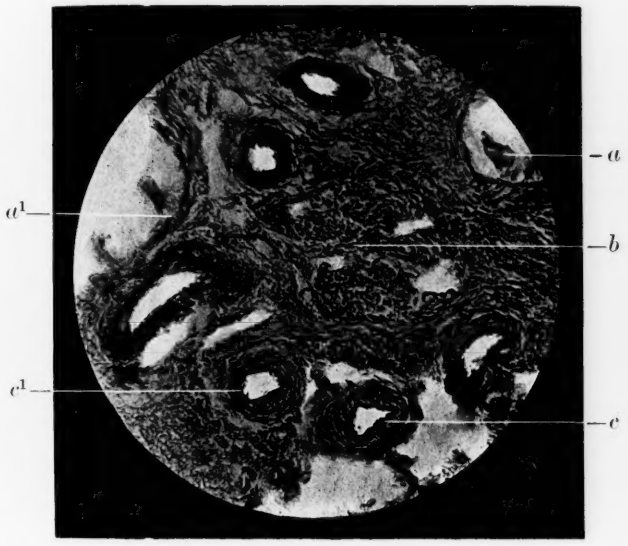

Fig. 12. Same case as above. Weigert. Small vessels with definite walls and well-marked internal elastic laminæ occurring within the endometrium. (× 200.)

a, a^1, Unstained glandular epithelium. *b*, Unstained stroma of mucosa. *c*, c^1, Inner elastic lamina.

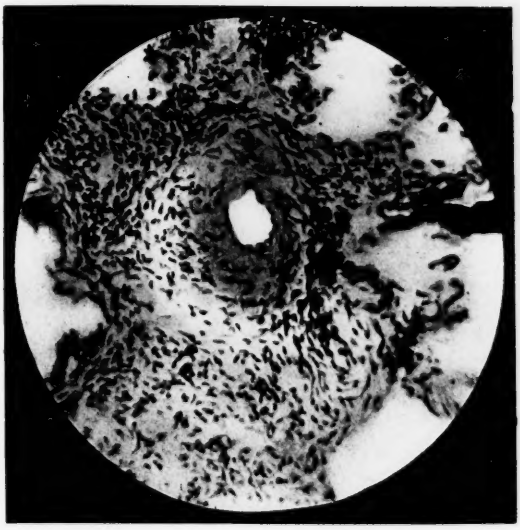

Fig. 13. Same case as above. "Hyaline" degeneration of an endometrial capillary. Note the circular arrangement of the stroma round the vessel. (× 230.)

here the number of dilated lymphatic spaces and the numerous groups of vessels with thickened walls are striking.

Muscular wall. The muscle appears on the whole well-preserved. There is no marked increase in the amount of fibrous tissue, but in some parts the fibrous tissue which throughout the specimen penetrates between the individual muscle fibres, appears to have increased and produced atrophy of the enclosed muscle fibres. No broad bands of fibrous tissue are to be seen in the specimen except such as form the adventitial coats of the larger vessels. The fibrous tissue throughout shows a tendency to degenerate.

Vessels. The larger vessels in the vascular zone show a marked thickening of all their coats. In the arteries, the intima as a rule is irregularly thickened, forming definite projections into the lumen in some cases. In sections stained by hæmatoxylin and eosin the thickened intima is seen to be poor in nuclei and more or less degenerated; with Van Gieson's stain there appears a considerable increase in fibrous tissue which is markedly degenerated; while Weigert's elastic stain shows a definite increase in elastic fibres throughout the intima in many of the larger arteries. This is strikingly seen in a branch of the uterine artery at its point of entrance to the uterine wall (Fig. 14). The media also in this vessel is greatly thickened, and while the muscle nuclei are fairly well preserved, as seen in a hæmatoxylin preparation, there are signs of atrophy of the muscle in parts. The muscle fibres are seen, in a Van Gieson preparation, to be interpenetrated by a degenerating fibrous tissue which apparently presses on and destroys the muscle cells. This is also to be seen in the arteries within the uterine wall, though to a much less extent; the usual condition is a hypertrophy of the media without evident degeneration. The adventitia is thickened and degenerated in the larger arteries, appearing in Van Gieson sections as a granular red or pink mass, and not taking Weigert's stain. The veins do not show much change from the normal. In some cases both coats are thickened, the inner showing irregular proliferation filling up the lumen, and the outer showing an increase in elastic tissue which also extends in fine fibrils through the inner coat. The smaller arteries show swelling of the endothelial cells as a rule, or slight irregular proliferation of the intima; slight thickening of the media; and marked thickening in the adventitia (Figs. 9 and 10). The groups of vessels so affected occurring amongst the glandular tissue at the junction of mucosa and muscle are particularly striking, (Fig. 11.)

Cervix. There is a cystic condition of some of the glands, and

here and there a subepithelial hæmorrhagic infiltration. The vessels are slightly thickened in all their coats, but there is no marked increase in the elastic tissue. The arteries retain their well-marked internal elastic lamina, which appears thickened only in one or two of the larger vessels.

It is noticeable that in this case there is no typical " periarterial " or " perivenous " degeneration. The degeneration here is in the fibrous tissue of the thickened adventitial coats, the degenerated areas looking pink and granular with Van Gieson's stain, and not staining at all with Weigert's. So far it might appear that there is a failure in the normal increase in elastic tissue. But, on the other hand, there is in the larger vessels particularly a definite increase in elastic tissue in the intima; and the uterus in this case had been only once pregnant. The possibility of infection must not be overlooked, since there was evidence of adnexal inflammation. Addinsell describes degeneration of the fibrous tissue as the third stage of the pathological process in " chronic infective metritis."

Certainly failure of increase in the elastic tissue will not explain the hæmorrhage in another case examined, that of a woman, aged 45, with a history of severe menorrhagia for ten years, and in whom death suddenly occurred three months after curetting from septic peritonitis evidently originating from an acute endometritis (Path. No. 2505). The vessels show marked and extensive degeneration within and around their walls (Fig. 4). The intima is not markedly affected, but the media in most of the larger arteries is very greatly thickened and degenerated; and in the most advanced cases both media and adventitia are represented in Weigert sections as solid clumps of elastic tissue (Fig. 5). The veins have thickened inner coats of well formed fibrous tissue, and show an increase in elastic tissue in their outer coats in the position occupied by degenerated areas of few nuclei in hæmatoxylin and eosin preparations. The vessel condition is practically identical with that found in the senile case already described, although the changes have not extended to the smaller vessels to the same extent. The musculature in this case is separated up by bands of fibrous tissue which is well-formed and not degenerated. The cause of the menorrhagia is not evident.

Is there, then, an arterio-sclerosis of the uterine vessels apart from that brought about by physiological causes? If so, will the condition necessarily be accompanied by hæmorrhage?

If we consider how the physiological changes are brought about, the question becomes clearer. Westphalen and Thoma have endeavoured to show that the peculiar vessel changes in the uterus are essentially

due to the changes in blood pressure to which the functionally active uterus is subject. The periodic increase in blood pressure at the menstrual periods, and still more the pressure changes incident to pregnancy and the puerperium, cause changes in the uterine vessels to which no other vessels in the body are subject to the same extent. The uterine vessels are, so to speak, shorter-lived than any others. If, now, it be supposed that changes in blood pressure in these arteries arise from some pathological condition, apart from pregnancy, etc., similar changes will presumably occur.

Theoretically, we may suppose that persistent high tension in the uterine and ovarian arteries will bring about as a first result, through increased vasomotor stimulation, an exaggerated response on the part of the musculature, so that a true "work-hypertrophy" takes place, *i.e.*, hypertrophy of the uterine muscle generally, including the muscular coats of the arteries. Since arteries acquire their coats in proportion to the pressure they are called on to resist, it is probable that the adventitia will be strengthened by an increase in fibrous tissue. The strain must necessarily tell on the intima. In all parous uteri the inner elastic lamina of the arteries is thickened. In the case described, where general arterio-sclerosis was present, the larger arteries showed a marked increase in elastic tissue in the intimal coats. Probably this is the nature of the compensatory change when the artery wall is subjected to a sustained increase in blood pressure. Later the continued pressure will bring about degenerative changes; in the intima, whose nuclei disappear to a great extent; and in the media, where fatty degeneration and atrophy of the muscle may occur. We may further suppose that if the increase in pressure be gradual and sustained, as would occur in a case associated with general arterio-sclerosis, the compensatory change will extend gradually to smaller and smaller branches of the vessels, reaching eventually the endometrial capillaries. When degenerative changes have occurred, and the vessels can no longer react to the increased blood pressure, in other words, when compensation is disturbed, hæmorrhage will result. The importance of menstruation in starting the hæmorrhage has already been seen.

All these changes are illustrated in the case described above, in which there was general arterio-sclerosis, and a fairly clear history of syphilitic infection dating back twenty years. The peculiar pathological features in the case are (1) the very distinct affection of the intima, which showed irregular proliferation and an increase in wavy elastic fibres out of all proportion to the general elastic increase, which was only very slightly marked, throughout the

uterus; (2) the marked fibrosis around the vessels, particularly the smaller vessels, unassociated with fibrosis of the uterus generally; (3) the occurrence of groups of thickened vessels at the junction of mucosa and muscle, and the tendency of these vessels to invade the mucosa; (4) the existence of vessels with definite walls and a well-marked internal elastic lamina within the mucosa, and thickening and degeneration of the "thick-walled" endometrial capillaries.

The case is clearly one of uterine arterio-sclerosis associated with general arterio-sclerosis. A clear case of pathological uterine arterio-sclerosis existing as a purely local condition has yet to be demonstrated.

The changes in the vessels due to physiological processes cannot be regarded as causes of hæmorrhage. They may go to an extreme degree without the occurrence of any abnormal bleeding. This is only natural. Further, since these changes are essentially brought about by the changes in blood pressure associated with the functionally active uterus, they are possibly indistinguishable from changes due to a pathological increase in blood pressure. In the causation of hæmorrhage, the important thing is the pathological increase in blood pressure which gives rise to the vessel condition. To speak of the arterio-sclerosis as the cause of the hæmorrhage is a mere confounding of words. The hæmorrhage is a symptom of the condition of which the arterio-sclerosis is a sign. The association between hyperpiesis and arterio-sclerosis in other parts of the body is well known though not clearly understood. It seems clear in some cases that the increased blood pressure precedes the change in the vessels and is the cause of it, although the case is complicated by the fact that the vessel change may itself induce a rise in blood pressure by increasing the peripheral resistance. The real difficulty is that the ætiology of arterio-sclerosis occurring anywhere in the body is still obscure. After all the well-established causes, including syphilis, alcoholism, various forms of poisoning, etc., have been excluded, there remains a class of unexplained cases arising independently of these causes in comparatively young people and associated in some way with an increased arterial tension. Similar conditions may arise in the uterus, possibly associated with some form of local toxæmia. But the essential point is the condition behind the vessel change. It is readily conceivable that if an increase in arterial pressure occurred suddenly, or occurred in a uterus of deficient musculature, hæmorrhage of a severe kind might occur before any marked changes in the vessels had had time to occur. Thus Simmonds' case of uncontrollable hæmorrhage showed a sclerosis

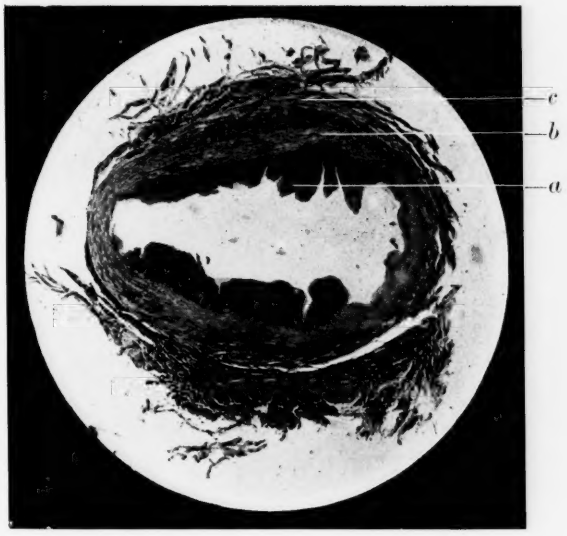

Fig. 14. Same case as above. Irregular proliferation of intima in a branch of uterine artery where it enters the uterus. Hæmatoxylin and eosin.

a, Intima. *b*, Media. *c*, Adventitia.

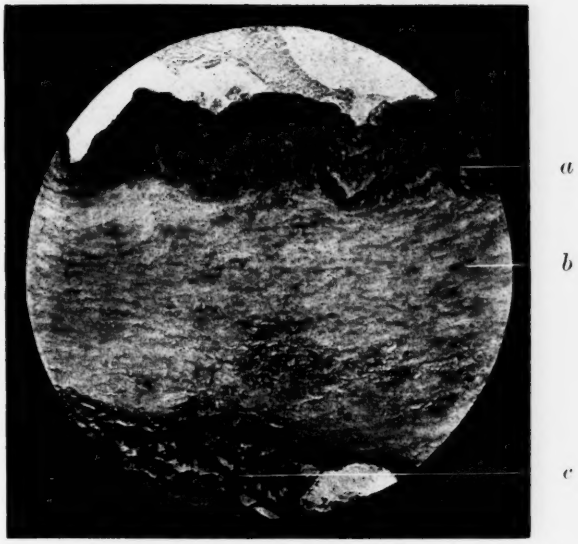

Fig. 15. Same artery as Fig. 14. Part of the wall to show the marked increase in wavy elastic fibres in the irregularly thickened intima. Weigert. (×100.)

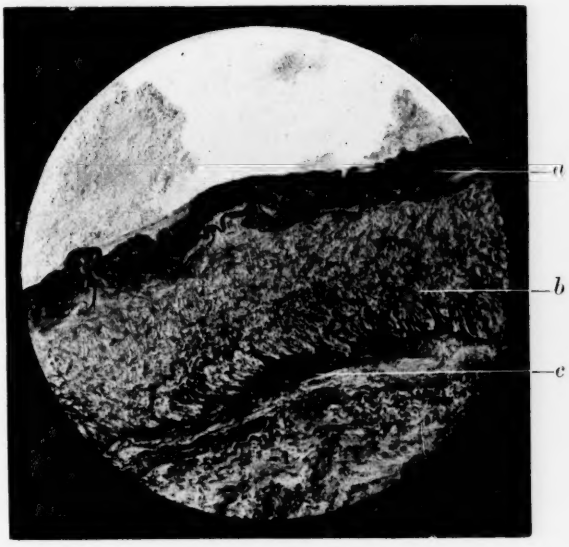

Fig. 16. Path. No. 4750. Senile case. Part of the wall of uterine artery to compare with Fig. 15. The intima is not so much thickened, and its elastic tissue is clumped together. Note the greater amount of elastic tissue throughout the media in this case as compared with previous figure. (×100.)

a, Intima. *b*, Media. *c*, Adventitia.

of the uterine artery alone. At the opposite extreme are those cases where the changes have extended to the vessels of the endometrium, and where the diagnosis can be made from curettings.

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SELECT CLINICAL REPORTS.

(Under this heading are recorded, singly or in groups, cases to which a special interest attaches either from their unusual character or from being, in a special sense, typical examples of their class.)

I.

A Case of Vesical Calculi Due to Perforation of the Bladder by a Suppurating Dermoid Tumour of the Ovary.

By J. A. C. Kynoch, M.B., F.R.C.S. (Edin.).
Professor of Obstetrics and Gynæcology, University of St. Andrews.

Fistulous communications between the bladder and surrounding organs—exclusive of the ordinary genital fistulæ—are not very common. The most frequent and perhaps most interesting cases are those where a suppurating dermoid ruptures into the bladder, with the possible subsequent formation of a vesical calculus. More rarely does the communication between cyst and bladder arise from an active mechanical boring process through the septum. Of the latter only three cases have been recorded, viz., those of Münch, Blich-Winge and Waille. Münch describes a case carefully examined post mortem, where a papillomatous growth resembling a raspberry and having its origin in a left-sided ovarian cyst equal in size to a hen's egg had penetrated through the posterior bladder wall.

Vesical calculi following the rupture of a dermoid cyst may have as their nuclei bone, hair or teeth. The following are the notes of a case showing the formation of a calculus around a lock of hair derived from a dermoid cyst of the left ovary:—

Mrs. E., æt. 42, was admitted to the gynæcological department of the Dundee Royal Infirmary on November 6, 1903. She complained of inability to retain her urine, which for a year she had noted to be blood-stained. The urine was highly ammoniacal, had a sp. gr. of 1026, and contained albumen, pus and phosphates in abundance. The patient's general condition was unsatisfactory.

Through the anterior fornix a hard, fixed swelling was felt, which on passing a catheter into the bladder proved to be a large calculus.

On November 11th two calculi weighing 1115 and 1065 grains respectively were removed by vaginal cystotomy. On washing out the bladder with boric acid solution, dermoid material was found

escaping from the urethra. Further, pelvic examination showed the presence of a tumour, equal in size to an orange, to the left of the uterus. The condition was now recognized to be a left-sided suppurating dermoid ovarian cyst, which had penetrated the bladder wall. The patient's weak condition absolutely contra-indicated immediate abdominal section. Her bladder was irrigated twice daily, and three weeks later, her general condition having improved, abdominal section was performed. The ovarian dermoid was found densely adherent to surrounding parts. The adhesion to the bladder wall was so firm as to necessitate resection and suture of the portion of the bladder wall involved. The patient died ten days later with all the symptoms of ascending kidney infection.

The accompanying photograph shows a section of one of the calculi, with a lock of hair in the centre forming the nucleus around which phosphatic deposit had collected.

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Section of Calculus with Lock of Hair as nucleus.

II.

Further Report of an Already Recorded Case of Co-existing Cancer of the Cervix Uteri, Cancer of the Corpus Uteri and Cancer of the Ovary.

By Walter W. H. Tate, M.D.

In Dr. Cullingworth's Ingleby lectures for 1904, published in this Journal for June and July of that year, there was related an interesting and unusual case in which there co-existed, without visible contact or continuity, carcinoma of the cervix uteri, carcinoma of the corpus uteri, and carcinoma of the right ovary. The case was still under observation at the time the lectures were delivered, and some signs of recurrence were observed even while they were passing through the press. It seems desirable therefore, that the after history of the case should be published, and as the patient has been under my observation from that time to the present, Dr. Cullingworth thought it would be desirable that I should place on record her present condition. Before doing so, it will be convenient, even at the cost of some repetition, to give the main particulars of the case as recorded in Dr. Cullingworth's second lecture. (See *Journ. of Obstet. and Gynæcol.*, July 1904, and *Birm. Med. Review*, July 1904.)

The patient was first seen by Dr. Cullingworth on the 27th of June 1901. She was then 33 years of age and unmarried. There had been, for about fourteen months, occasional menorrhagia, and an intermenstrual discharge pink in colour. She had not hitherto been examined internally. The cervix uteri was found to be long and patulous, the finger passing easily into what appeared to be the uterine cavity, where there was a growth easily made to bleed and easily detached. Behind the uterus, pushing it forwards and to the left, was an irregular fixed swelling equal in size to an ordinary orange. Dr. Cullingworth thought it probable that both the intra- and extra-uterine growths were malignant, but considered it impossible at the moment to make an absolutely certain diagnosis. He recommended exploration of the uterine cavity, with either curetting, or vaginal hysterectomy, or abdominal section as might be found necessary.

On July 10th, 1901, the patient was anæsthetized. "The cervical canal was found curiously and asymmetrically dilated. The os uteri internum was situated in the left upper corner of the expanded cervix, on the wall of the upper part of which the mucous membrane was covered with a soft growth. The os internum was sufficiently widely open to admit the finger. Some soft growth was felt on the lining membrane of the fundus similar to that found in the upper part of the cervix. (See drawing.) This was removed with the curette and a portion sent for microscopical examination to the Clinical Research Association. On introducing the finger into the uterus after the curetting, a hard, smooth, raised plaque, equal in size to an almond, was felt just beyond the os internum. As the material removed by the curette had not, to the unassisted eye, a malignant appearance, it was decided to leave the uterus *in situ* until the microscopist's report had been received, and to proceed to abdominal section with the view of removing, if possible, the intra-pelvic swelling, which was now, under anæsthesia, felt to be equal in size to that of a fœtal head at term. The tumour proved to be a thin-walled multilocular cystic adenoma of the right ovary, one loculus of which was ruptured during removal. The tumour was adherent to the floor of the pelvis, to the lower part of the posterior aspect of the uterus, and to the back of the broad ligament. It measured about 5in. by 4in. At one spot there was a small projecting mass about the size of a small walnut, consisting of a compact mass of very small cysts, which gave it a solid appearance. There was some papilloma on the inner surface of the unruptured larger cyst. (Portions of both were sent for examination and report.) The tumour, having been separated and drawn out, presented a good pedicle of stretched out broad ligament. When it had been removed, the uterus was brought into view, and was found globular and bulky, like a two-months pregnant uterus, except that it was very firm. The left ovary was small, shrivelled, adherent, and calcareous. It was not removed. On the 20th of July, ten days after the operation, a report was received from the Clinical Research Association, signed by Mr. Targett, to the effect that the disease both in the ovary and in the uterus was carcinoma.

"The following is a copy of Mr. Targett's report:—

'*Specimen 541.*—The material from the uterus consists of a very soft columnar-celled carcinoma of the villous type. The tubular arrangement of the cells is well preserved, and the stroma is very scanty.

Sketch showing (*a*) the growth at the fundus uteri; (*b*) the apparently independent growth at the upper part of the widely-dilated cervical canal. (From a drawing kindly made by Dr. C. Hubert Roberts.)

'*Specimen 542.*—The character of the carcinoma is best seen in this section from the right ovary. It shows a typical columnar-celled growth, and a marked tendency to the formation of small cysts which become secondarily filled with intra-cystic papillary processes. There is abundant evidence of malignancy in the invasion of the surrounding stroma.

'*Specimen 543.*—This section at first sight seems to be a simple adenomatous intra-cystic growth, but on closer inspection it will be noted that there are many solid clumps of epithelium among the delicate papillomata. They are due to epithelial proliferation and indicate the tendency of the tumour, though here seen in its earliest stages.

'Apparently the disease is primary in the ovary and has extended to the uterus.

J. H. TARGETT.'

'*July 19th, 1901.*

"The patient was therefore advised to have the uterus extirpated *per vaginam*, and to this she consented.

"Accordingly, on the 25th of July, vaginal hysterectomy was performed by my colleague, Dr. Walter Tate. The operation presented no special difficulty. There was a distinct hard lump, about the size of a Spanish nut and rough on its inner surface, situated in the anterior wall, towards the right side. There was no evidence of extension of the disease to the broad ligaments or to the left appendages. The latter were, however, removed as a matter of precaution. The parts in the neighbourhood of the stump on the right side were adherent, but the adhesions were easily separated. The patient recovered fairly well from the operation, but remained for some months pale, thin, and thoroughly unstrung. She spent the winter of 1902–3 on the Italian Riviera, and whilst there underwent the Weir Mitchell treatment. The result was successful beyond anticipation, the patient gaining 22lbs. in weight during the six weeks. She returned to England looking bright and happy and the very picture of health. In the summer of 1903, about two years after her operations, she was married, her husband having been made fully aware of all the circumstances. Vaginal examinations had been made frequently ever since the last operation, but with entirely negative results until the 24th of November, 1903, when a little fleshy growth was detected at one angle of the scar in the vaginal roof. There had been a slight continuous blood-stained discharge for some time, and the little growth bled easily on touch. I advised that this should be at once dealt with. I accordingly sent the patient to Dr. Tate, who

regarded it as merely a little exuberant granulation-tissue, but quite agreed with me that it should be removed. This was done on the following day, and the specimen was sent to the Laboratory of Pathology for examination. The report was to the effect that the growth was distinctly carcinomatous."

On the 17th March, 1904, Dr. Cullingworth and I found a soft swelling above the vaginal roof equal in size to a pigeon's agg. Two days later I made an exploratory incision in the vaginal roof with the result of evacuating a small collection of discoloured serum. The swelling thereupon disappeared. No evidence of new growth was at that time detected.

In June of the same year, the patient was again found to have a swelling, this time equal in size to a closed fist, above the vaginal roof. The swelling was discovered accidentally in the course of an ordinary periodical examination. Except that the patient had felt somewhat lacking in energy, and had lost flesh slightly, she had had no symptoms. Dr. Cullingworth advised that the abdomen should be re-opened and the swelling, if possible, removed. Accordingly, on June the 20th, 1904, I performed an exploratory operation. The swelling proved to be a cyst connected with the remains of the right uterine appendages, roofed in by adherent viscera and itself intimately adherent to the parts around. It contained reddish-brown serum without odour. Its wall, for the most part thin, smooth and friable, presented at one spot a patch of soft, friable growth, equal in size to a hazel nut. The whole of the cyst, including the growth, was removed. No evidence of further extension or of glandular invasion could be detected.

The growth from the cyst-wall was examined microscopically and pronounced to be malignant papilloma.

The patient made an excellent recovery.

She has continued to present herself for examination at regular intervals, and no evidence of recurrence has hitherto been detected. The last examination took place on the 20th of November, 1906, with an absolutely negative result. As it is now two years and a half since the last operation, the prognosis may, I think, be considered favourable. The patient's general health is very good. She is becoming stouter and is enjoying her life and in excellent spirits.

Remarks. The case is a very remarkable one, and seemed quite worth placing on record in some detail. The non-recurrence of the growth removed in November, 1903, from the angle of the vaginal incision, and pronounced by the microscopist to be carcinomatous, is

to my mind one of the most surprising features of the case. Of course, it may be said that the disease may even yet make its re-appearance, but in a comparatively young patient, where one would expect any fresh developments to take place rapidly, a freedom from recurrence of $2\frac{1}{2}$ years' duration certainly entitles one to hope that a cure has been effected. And if that be so, the case is a most valuable testimony to the benefit that may result from perseveringly following up such cases and dealing promptly with every apparent recurrence of the growth.

Note by Dr. Cullingworth.

In the other of the two cases of concurrent carcinoma of the uterus and ovary mentioned by me in my Ingleby Lectures, the sequel was less satisfactory. The operations had in that case taken place fourteen months before the date of the Lectures, and up to that time (May 1904) there had been no definite recurrence, though some thickening "such as might be accounted for by matted adhesions" could be felt between bladder and rectum. Meantime the patient had been able to go about paying visits and generally to lead a fairly active life. Some four or five months later, however, viz. in Sept. and Oct. 1904, unmistakable signs of recurrence manifested themselves in a form that precluded surgical relief, and the patient died on the 7th of Dec. 1905, having survived the operation for 21 months.

In this case, however, it may be remembered that much valuable time was lost before the patient could be induced to consent to an operation.

REVIEW OF CURRENT LITERATURE.

UTERUS.

The Lower Uterine Segment.

ASCHOFF (L.). *Zeits. für Geburts. und Gynäkol.*, 1906. Bd. lviii., Heft 2.

THE lower uterine segment was the subject of a spirited discussion at the Berlin Obstetrical and Gynæcological Society in December, 1905, and March, 1906. Aschoff considers that debate on the subject is useless until the meaning of the terms "cervix" and "lower uterine segment" has been agreed upon. The macroscopic designation of the cervix does not correspond to the microscopic. Macroscopically the cervix extends from the external os to the narrowest part of the uterine cavity. Its upper limit is marked by a slight constriction externally, by the line of attachment of the peritoneum, by the entrance of the large vessels, especially by the circular vein, and corresponds to about the middle of the virgin uterus. This upper limit is the internal os, for which Aschoff suggests the name "*orificium internum anatomicum.*" The upper part of this "macroscopic cervical canal" is clothed by mucous membrane like that of the body of the uterus, the only difference being that the mucous membrane which clothes the upper third of the cervical canal forms deeper and less regular indentations in the musculature. The uterus must then be divided into—(1) the cavity of the uterus, lined by true corporeal endometrium; (2) the upper third of the cervical canal or isthmus, lined by endometrium like that of the body; and (3) the cavity of the cervix, the lower two-thirds of the cervical canal, slightly spindle-shaped, lined by typical cervical endometrium. Between (1) and (2) is the *orificium internum anatomicum*, or internal orifice of the uterus. Between (2) and (3) is the *orificium internum histologicum*, or internal orifice of the cervix. This classification alone gives the key to the true explanation of the lower uterine segment—the lower uterine segment is the isthmus. The isthmus during pregnancy transforms itself into the lower uterine segment, on which the ovum may be implanted. The upper limit of the lower uterine segment, the so-called contraction ring, corresponds to the internal orifice of the uterus, the region of the circular vein and of the attachment of the peritoneum; the lower limit lies, in the majority of cases at least, just above the internal orifice of the cervix, *i.e.*, the junction between the corporeal and cervical endometrium. The opening up of the cervical cavity, to which attention has often been drawn, has nothing to do with the formation of the lower uterine segment.

HENRY RUSSELL ANDREWS.

The Nerves in the Substance of the Uterus.

LABHARDT. *Archiv für Gynäkol.* Band lxxx., Heft 1, p. 135.

LABHARDT found an extensive system of nerves in the uterus of the rabbit and in the human uterus. The main nerve trunks run between the two middle layers of the uterine muscles, and give off intra-fascicular branches. The termination of the nerve fibres could not be determined by the methods employed (Golgi and methylene blue), and ganglion cells could not be detected. The author believes that uterine contractions are brought about—(*a*) (neurogenically) by stimulation of the ganglion cervicale or by a reflex; (*b*) (myogenically) by direct stimulation of the muscle.

H. T. H.

The Motor and Inhibitory Nerves of the Uterus.

FELLNER. *Archiv für Gynäkol.*, Band lxxx., Heft 2.

THE author publishes a new series of experiments on the innervation of the uterus, and comes to the following conclusions:—The nervi erigentes are motor nerves for the longitudinal muscles of the uterus and circular muscles of the cervix, and inhibitory nerves for the circular muscles of the uterus and the longitudinal muscles of the cervix. The hypogastric nerves are motor nerves for the circular muscles of the corpus uteri and the longitudinal muscles of the cervix, and inhibitory nerves for the longitudinal muscles of the uterus and the circular muscles of the cervix. The muscles of the vagina are acted upon in the same way as the uterine body. The author illustrates his paper with curves from the actual experiments.

H. T. H.

Investigations on the Coagulation of the Blood during Menstruation.

BIRNBAUM and OSTEN. *Archiv für Gynäkol.* Band lxxx., Heft 2.

THE authors record their experiments on the blood of menstruating women which demonstrate that in nearly every case coagulation is retarded. The experiments were made by adding small quantities of a solution of fibrinogen (Morawitz) to the menstrual blood-serum. The cause of the defective power of coagulation is not known, but the authors say that their experiments discredit the old doctrine that the alkaline mucus in the cervical canal prevents the coagulation of the menstrual blood.

H. T. H.

Absence of the Uterus in Three Sisters and in Two Cousins.

BOSTON. *Lancet*, 1907. Vol. i., p. 21.

THE vagina, labia and clitoris were normal in these cases, but no evidence of cervix or body of uterus could be found on bimanual examination or by the speculum. The two cousins have apparently nothing to do with the three sisters, and the author's information about them is second-hand. Similar cases of this malformation in two sisters, recorded by Phillips, and in three sisters out of a family of five, recorded by Sedgwick, are also referred to.

J. S. F.

The Anatomy of Exfoliative Menstrual Endometritis.

ASCHHEIM. *Archiv für Gynäkol.* Band lxxx., Heft 2.

THE author believes that the membranes found in dysmenorrhœa are due to an exudative endometritis, present at the time of menstruation. The membranes vary a great deal in structure. They mostly show fibrinous matter, stroma cells, glands and remains of mucous membrane; decidual cells have been found. Virchow's nomenclature "decidua menstrualis" is of course correct, "dysmenorrhœa membranacea" is not suitable, as the periods are sometimes painless; but "endometritis exfoliativa" is, anatomically speaking, the best name for this condition. The ætiology of the disease is not known. Heredity seems to play a part. Pain may be intense, but is not a constant symptom. Sterility is frequently complained of. The prognosis of a lasting improvement is bad. Relief may be obtained from curettage, hydrotherapeutics and drugs, such as hydrastis, ergot, etc., but no definite cure can be expected until the ætiology of the disease is known. H. T. H.

The Sclerosis of the Uterine and Ovarian Vessels during Gravidity, Menstruation and Ovulation.

PANKOW. *Archiv für Gynäkol.* Band lxxx., Heft 2.

PANKOW'S investigations lead him to state that gravidity produces sclerosing changes in the vessels of the inner and chiefly in the submucous coats of the uterus; that menstruation causes sclerosing changes in the vessels of the mucous layer, and that an ovulation sclerosis has been observed in the ovarian vessels. These changes occur in the small vessels mainly, whereas senile sclerosis preferably attacks the larger vessels. The author appends a table of diagrams illustrating the conditions found. H. T. H.

The Treatment of Uterine Displacements.

PORTER MATHEW. *Transvaal Med. Journal*, 1906. Vol. ii., No. 3, p. 56.

THIS paper contains a very full résumé of recent teaching on this subject, and is interesting as showing the extraordinary diversity of opinion which exists as to the importance of uterine displacements. In concluding, the writer says: ". . . . a brilliant operation is not necessarily good surgery. Even apart from mortality no surgeon can boast of uninterrupted success. The personal equation must enter largely into our decisions. Nor must we fail to duly weigh as factors our patient's environment, mode of life and temperament." J. S. F.

Clinical Contribution to the Calcification of the Myoma of the Uterus.

GRÜNBAUM. *Archiv für Gynäkol.* Band lxxx., H. 1, p. 212.

THE author is anxious to contradict a still current belief that calcification is a desirable form of degeneration in a uterine myoma. He publishes two cases, in which he attributes the determining cause of the fatal result

to calcification. In Case i. the tumour was invested with a calcified shell ½ cm. thick, which had to be sawn through to get at the interior, which was composed of ordinary fibromyomatous tissue. The omentum was adherent to the front of the tumour, and was converted into a hard plate. The patient was only 22 years old; as a rule these calcifying processes are not met with until the 40th or even the 50th year, and commonly start from the centre and extend to the periphery, and not *vice versâ*, as in this case. Firm adhesions to bladder, omentum, intestines, etc., are typical of a calcified myoma and extremely rare in ordinary fibro-myomata. The second patient was 62 years old. The tumour, which had been present for 10 years, had only begun to give trouble a few weeks before operation. In this case the tumour had to be drilled open before it could be pulled forward, and had to be chipped away in little bits. It was more uniformly calcified, of an ivory colour and consistency, the bony network being interspersed with ordinary fibro-myomatous material. Calcified myomata are apt to produce severe pressure symptoms, viz., pain and constipation, which may lead to absolute obstruction, perforation of the bowel and bladder, etc., and also to œdema of the lower extremities, hydronephrosis, etc. The chemical composition of these two calcified myomata was: calcium phosphate 62 per cent., calcium carbonate 8·5 per cent., calcium sulphate 1·5 per cent. In a few recorded cases calcium carbonate was predominant. The characteristic feature of calcification is the abnormal ratio between the non-increasing size and the increasing symptoms, and also a tendency to sepsis and putrid discharges. Early operation is advised as the difficulties increase enormously with progressing calcification. Photographs of the two tumours are included in Grünbaum's interesting paper. H. T. Hicks.

Intra-peritoneal Bleeding from a Uterine Fibroid, with Acute Distension of the Abdomen.

Clarke (Bruce). *Lancet*, 1907. Vol. i., p. 8.

A case of this nature is recorded, the diagnosis before operation being perforation of a gastric or duodenal ulcer; the hæmorrhage appeared to be due to ulceration of vessels on the surface of the tumour from pressure on the sacral promontory. A brief review of similar cases is given. The greater part of the paper is occupied with a very interesting account of the treatment of the acute abdominal distension by enterostomy and of the means by which the patient was kept alive by feeding the lower end of the fistulous bowel by the fluid which escaped from the upper end. The patient recovered after the enterostomy had been closed by an end-to-end anastomosis. J. S. F.

The Functions of the Ovaries after Total Extirpation of the Uterus, and their Relation to Post-operative Manifestations.

Holzbach. *Archiv für Gynäkol.* Band lxxx., Heft 2.

Judging from his own investigations and from the records of published cases, Holzbach finds that the ovaries retain their full power of ovulation even many years after total removal of the uterus. If, nevertheless, manifestations of disturbance of function occur in these patients, the

author believes that the cause must not be looked for in the ovaries only. Doubtless there are extensive and delicate nerve relations between the individual organs of the female genital tract which may be responsible for the causation of such disturbances. The author concludes by urging that surgeons should endeavour to be as conservative as possible in their methods of operating on cases in which the removal of the uterus is indicated, especially in operations for myomata.

H. T. H.

Operations for Recurrent Carcinoma of the Uterus.

FRANZ. *Archiv für Gynäkol.* Band lxxx., Heft 2.

FRANZ has performed 16 operations on 12 women for recurrent carcinoma of the uterus with one death; seven patients were discharged healed, free from discomfort and apparently well, three were discharged with a vesico-vaginal fistula, and one with a recto-vaginal and a vesico-vaginal fistula. The author thinks that these results are sufficiently encouraging to recommend operation in every case of recurrent carcinoma of the uterus except in those where the metastases implicate the large vessels.

H. T. H.

Amputation of the Cervix with the aid of an Intra-Uterine Tenaculum.

COFFEY (R. C.). *Surgery, Gynæcology and Obstetrics,* December, 1906.

To draw down and steady the cervix the author uses a cat-claw tenaculum. The tenaculum consists of a tube with a couple of pairs of eyes in it and a central rod with two pairs of claws which lie concealed in the tube until it is inserted. The central rod is then pushed in and the claws protrude at the eyes and penetrate the anterior and posterior walls of the cervical canal. The amputation is then done with the tenaculum in position. When the operation is finished the claws are sheathed by drawing back the central rod and the instrument is removed.

ROBERT JARDINE.

VARIOUS.

Fibroma and Fibro-myoma of the Mesentery in Women.

BOWERS. *Annals of Surgery,* December, 1906.

BOWERS, of Dayton, Ohio, U.S.A., reports a case of fibroma of the mesentery in a young girl, and adds abstract notes of 23 cases already published. Out of the 24 no fewer than 18, or three-quarters, occurred in women. Bowers's own patient was a factory girl, aged 15, who missed a period, suffered from nausea in the evening or afternoon, and at the end of a few months noticed an enlargement on the left side of the abdomen on a level with the umbilicus. When examined under chloroform the swelling could not be detected, but it once more became palpable when the patient

recovered her consciousness. This phenomenon again occurred when Bowers himself used an anæsthetic. He made an exploratory incision, and found the tumour in the lesser omental cavity, attached by a short pedicle to the transverse meso-colon. There were firm adhesions to the posterior wall of the stomach. The tumour apparently fluctuated, but proved to be solid when punctured. After enucleation and separation from adhesions the tumour was set free by ligature and division of its pedicle. As usual, there was much hæmorrhage; in order to check it the cavity whence the tumour had been enucleated was packed with gauze wrung out in hot saline solution. After firm pressure for five minutes bleeding vessels were more easily detected and secured, but it was found necessary to pack the cavity with gauze once more. Half the gauze was removed at the end of twelve hours, the remainder twelve hours later. When the operation itself was completed shock was so marked that transfusion and stimulation were found necessary, but the patient soon revived and made a good recovery. The tumour was a soft fibroma, made up of flattened or spindle-shaped cells of fibrillar connective tissue, with very irregular arrangement of intercellular substance. It measured 4 inches in long diameter, 3 inches in width, and over 1 inch in thickness.

Bowers adds abstract notes of 23 cases compiled from the tables already prepared by Bégouin (*Revue de Chirurgie*, Vols. xviii. and xix.), the reporter (*British Med. Journ.*, Vol. ii., 1904, p. 1075), and, more recently, by Vance ("Solid Tumours of the Mesentery, with Report of a Case and a Review of the Literature." *Annals of Surgery*, Vol. xliii., March, 1906, p. 366). The reason for the greater frequency of this kind of tumour in women is not explained by any of the above-named writers; it has been suggested that men are more likely to overlook an abdominal tumour of this kind, but as it has often been known to attain a large size in male patients and nearly always causes them discomfort and dyspeptic symptoms, this theory is not highly probable.

ALBAN DORAN.

Retroperitoneal Perirenal Lipoma.

REYNOLDS (EDWARD) and WADSWORTH (RICHARD G.). *Annals of Surgery*, July, 1906.

THE authors have compiled a valuable study of large retroperitoneal lipomata of perirenal origin. They dwell on the measures which should be taken for their safe removal based on anatomical studies, and report a case under their personal observation. Wadsworth, after making a careful dissection of a normal subject, concluded that the perirenal space, in which is contained the perirenal fat and to which a tumour growing from this fat must be limited, is bounded in front by the prerenal fascia and behind by Gerota's retrorenal fascia. The latter lies behind the perirenal fat, kidneys, ureter, and renal vessels in direct apposition to the posterior abdominal wall, and is of no particular surgical importance. The perirenal fascia, on the other hand, lies in front of the perirenal fat, kidneys, ureter, and renal vessels, and is of extreme surgical importance, since it separates these organs from the other vital structures in the immediate neighbourhood.

The patient under the authors' observation was a single lady, aged 38, who had consulted her doctor for neurasthenia and indigestion, with a

sensation of weight in the abdomen for eight months. These symptoms were associated with increase of girth, so uniform that she had not considered it important. On examination, percussion was found to be dull in the right iliac region and uniformly flat elsewhere except in the epigastrium, where the tympanitic note was separated from the flat by an almost straight transverse line. The uterus lay rather far forwards behind the pubes; there was no bulging of the vaginal roof, but on pressing the parietes firmly when the patient was under anæsthesia, a smooth, elastic, convex mass could be brought within reach of the finger in the vagina. It was diagnosed as a large new growth, probably ovarian, though unusually high for a tumour of that class, and possibly malignant.

On opening the abdomen a little to the left of the median line below the umbilicus, a large mass presented which occupied the entire left half of the abdomen, extending slightly beyond the median line to the right. It ended abruptly in a rounded extremity just above the pelvic brim. One of the longitudinal bands of the colon was visible on its surface, so that it was at first taken for an enormously distended colon. This error was dispelled when the rectal tube was passed into the bowel. The incision being enlarged till it reached an entire length of eight inches, the peritoneum became visible on each side of the colon, so that the tumour was at last recognized as retroperitoneal. It extended upwards higher than the hand could pass. The distended mesenteric vessels were clearly seen on the surface of the tumour. The peritoneum was opened by a transverse incision to the left of the tumour, and therefore external to the colon. This incision ran between parallel subperitoneal vessels, which were thus saved from injury. The tumour was now (after puncture) found to be a well encapsuled lipoma of great size. The capsule was divided to the extent of the transverse incision in the contiguous peritoneum. The hand was then cautiously inserted between the capsule and the surface of the fat, but as it was passed deeply the tension on the capsule became dangerous. Therefore the fat near the incision was drawn out and cut off with a knife, the detachment of the capsule continued, and then more fat cut away. At length enough space was gained to permit of the removal of the whole lower part of the tumour, the left kidney being reached by the operator's hand. As it became evident that the tumour was lobulated throughout, the later stage of its extraction consisted in separating lobule from lobule by gentle dissection with the fingers in the lines of cleavage. Great care was needed during the separation of the fatty lobes from the kidney, ureter and renal vessels; in this region the adipose tissue which made up the new growth was of the consistence and appearance of perirenal fat. The operator's hand could now be passed along the tumour across the median line above the root of the mesentery. The fat was here carefully separated from the aorta and vena cava and their branches; it became continuous on the right side, as on the left, with the perirenal fat. These connections were separated; the tumour ended in a mass which turned round the root of the mesentery and extended downwards in its capsule to some inches below the lower pole of the right kidney. The separation and delivery of the right extremity of the tumour did not, however, prove difficult. There was absolutely no hæmorrhage throughout the operation, and the only vessels which were tied were a large artery and vein which entered the tumour near the median line. A strip of gauze was placed in the empty capsule and brought out of the upper end of the abdominal incision, the remainder of which was closed with through and through silkworm-gut sutures. A continuous chromicized catgut suture

was applied to the peritoneum. The entire tumour filled a large china wash-hand basin, and weighed 14 lb. 14 oz. A great deal of bloody serum was discharged during the first twenty-four hours, when shock was marked; the gauze was removed on the second day, and the patient made a good recovery. It is instructive to note that she has since been troubled with neurasthenia and indigestion, as before the operation, although the abdomen has been relieved of nearly fifteen pounds of encapsuled fat pressing upon several highly-important structures.

[The authors refer to the reporter's case ("Retroperitoneal Lipoma weighing Thirteen Pounds Twelve Ounces," JOURNAL, Vol. ii., p. 244). There were strong adhesions of indurated fat connecting the tumour with the pancreas; the right kidney lay abnormally low. Drs. Reynolds and Wadsworth quote G. B. Johnson, who, in 1904, collected a total of 49 cases of retroperitoneal lipoma assumed to be of perirenal origin; Ahren, of Quebec, has recently published a fiftieth case. In only 3 was sarcomatous degeneration detected, and in only 1 was recurrence noted. We may, in respect to malignancy, refer to Markowsky's case of sarcomatous fibro-lipoma (*Annales de Gynéc. et d'Obstét.*,* August, 1905, p. 514). Ott, who operated, resected the cæcum and ascending colon, and was compelled to remove the right kidney, though it was normal. Death from shock occurred half an hour after the operation.]

ALBAN DORAN.

Cysts of the Urachus.

WEISER (W. R.). *Annals of Surgery*, October, 1906.

WEISER begins a short monograph on this subject by observing that both Wutz and the writer of this abstract were very cautious about the diagnosis of cyst of the urachus, so that their conclusions were destructive rather than constructive. Taking up an opposite standpoint, Weiser has prepared instructive tables of 64 cases reported as urachal cysts, including all Lawson Tait's well-known examples of "allantoic cysts." [The late Mr. Tait admitted that his theory might be wrong, although he disagreed with the reporter's views. Mr. Bland-Sutton was not positive about the interpretation of Tait's cases, and Byron Robinson, a recognized authority on the relations of the peritoneum, who assisted Tait at several of the operations on these disputed cases, admits that we have as yet no conclusive scientific post mortem evidence that the cysts were of urachal origin.]

Weiser reports 3 new cases, making 89 when added to those in his statistical tables. In 2 of Weiser's the patient was a female, and the two were almost at the opposite extremes of life.

CASE I. A woman, aged 75, had suffered for fifteen years from discharge of pus from the umbilicus, which became profuse. She was very stout, but Weiser could define a tumour of the size of a cocoa-nut in the median line between the umbilicus and the pubes. It could be raised

*The relative frequency of this class of tumour in the female, according to reports, is well-known. The same, I find, is the case with solid retroperitoneal tumours. Out of 13 which I have tabulated, 10 occcurred in women, and all the 13 were fibromata or fibro-myomata, removed by operation ("Fibro-myoma of Mesentery, 30 lb.: Removal: Recovery," *Brit. Med. Journ.*, Vol. ii., 1904, p. 1075). For evident reasons, however, there are sources of fallacy in estimating the proportion of cases in each sex from clinical records.

with the abdominal wall, to which it was apparently attached. Weiser, after excising the umbilicus and surrounding integuments, discovered a cyst-wall which bulged forwards under a deep layer of fat. Five ounces of pus came away, followed by a calculus which weighed seventy grains. The cyst had a thick and indurated wall, and dipped deeply down into the pelvis. It was washed out and packed with gauze; then the peritoneal cavity was opened above the upper limits of the cyst. Below, the cyst proved to be intimately connected with the bladder. The intestines being firmly adherent behind, only a small portion of the cyst-wall could be excised. A sinus developed which closed at the end of three months.

Case II. A girl, aged 11, was admitted into hospital after suffering for a few days from headache, vomiting and fever, with abdominal pain. The abdomen became distended and very tender. There was impaired resonance between the umbilicus and pubes, which extended to the right of the median line to about two inches, whilst on the left side it reached the loin. The skin around the umbilicus was deeply inflamed as though it lay over an abscess cavity. The diagnosis was obscure; tuberculous peritonitis (very reasonably) and suppurating urachal cyst were both held to be probable. When the abdominal incision was made, the parietal peritoneum was found to lie posterior to a cystic cavity, confirming the diagnosis of urachal cyst. The cyst-wall divided the abdominal cavity into two compartments, one, which almost filled the left side of the abdomen below the umbilicus, being the cavity of the cyst, whilst the other was the peritoneal cavity, occupying the right side. Except at one point, where the cyst-wall had ruptured, the collection of pus was extra-peritoneal. Inferiorly the cavity communicated with a duct which proved to be the lower part of the urachus patulous down to within three-eighths of an inch of the bladder. The urachus was divided at that point; then as much of the sac was taken away as could be dissected out without tearing through the wall. The cavity was washed out and drained with iodoform gauze. The patient apparently did well.

Case III. The patient was a man, aged 73. For six months he had been subject to abdominal pain, and there was a history of recent orchitis. Urine escaped from a small opening two inches below the umbilicus which had developed spontaneously in the course of the present illness. It continued to escape whenever the patient stood upright, and was entirely free from pus. The peritoneal cavity was laid open above the opening, then a sac was found anterior to the parietal peritoneum. This sac extended upwards to within an inch of the umbilicus and downwards into the pelvic cavity. Below, it was intimately associated with the bladder; posteriorly, the intestines adhered to its wall. After freeing as much of the anterior wall of the sac as was sufficient for the purpose, Weiser closed the urinary sinus by suture. A catheter was passed into the bladder through the urethra and retained for a few days, and the abdominal incision was closed without drainage. A urinary fistula developed two months later, but afterwards closed spontaneously.

The *tables* in Weiser's monograph include 28 cases not previously published. The author admits that the history of many of these cases is deficient and that some are of but small clinical importance. [A few have escaped Weiser's careful researches:—(1) Bryant. A woman was supposed to be suffering from an ovarian cyst, but the parietal peritoneum was found to be reflected from above the umbilicus on to the posterior part of the tumour, which was closely connected with the bladder. (2) Bryant. Patient a man aged 35. Bryant's two cases are published, as original

reports, in an appendix to the reporter's "Cysts of the Urachus; with Notes on Urachal and so-called 'Allantoic Cysts,'" *Med. Chir. Trans.*, Vol. lxxxi., p. 301. (3) Unterberger (*Monatsschr. f. Geb. u. Gyn.*, March, 1900, in a report of a meeting of a medical society). Retroversion of a gravid uterus was complicated by what was taken for rupture of an ovarian cyst through the umbilicus. Simultaneous passage of catheters through the urethra and umbilicus proved the existence of a cystic urachus and dilated bladder. Possibly, however, the canal of the urachus itself had not undergone cystic dilatation, although it must have become patulous; the influence of the uterine displacement is easy to understand. (4) Opitz. A small cyst of the urachus was detected during an exploratory operation. At first sight it was mistaken for a structure now of world-wide reputation, the vermiform appendix (*Zentralbl. f. Gynäk.*, No. 27, 1905, p. 855).—A.D.]

We find, on scrutinizing these tables, that in a total of 86 cases, 56 occurred in female patients and 20 in males, whilst in 10 there is no note about sex; in the analysis "7" is printed in error for "10." Adding Weiser's own cases, the total, as reported in females, is 58, and in males 21. Urachal cysts seem to be most common, or most frequently recognized, in middle life, 34 of the patients being between twenty and forty years of age, including 29 females. Of 15 who were past forty, 10 were females, and 5 males; whilst out of 14 under fifteen years of age an equal number, 7, were males and females. The tables also, we must add, include no fewer than 7 girls between the ages of sixteen and eighteen, 5 of the cases occurring in the practice of Lawson Tait; this reminds us that tuberculous peritonitis, and therefore encysted dropsy, is not rare amongst young girls. We may also learn, from a close study of the original reports so carefully tabulated by Weiser, that urachal cysts would be yet rarer in the male were it not that phimosis and stricture of the urethra as well as other diseases causing difficulty in micturition may force open Wutz's valve between the bladder and urachus. A urachal cyst may thus develop, although complete urachal fistula is probably more usual, especially in male infants and in boys.

The most constant factor, Weiser observes, in the *clinical history* of this condition is a mass between the umbilicus and pubes, which, however, varies greatly in size in different cases. The symptoms usually present are a feeling of illness, loss of flesh, pain, fever, gastric disturbance, *and the appearance of a tubercular subject.* Pus had been observed to escape from a fistulous aperture in the umbilicus in 15 per cent. of the cases when medical aid was first sought. The presence of polygonal epithelium in the sediment of the fluid is *said* to be pathognomonic.

Turning to *diagnosis*, Weiser observes that urachal cyst has been mistaken for tuberculous peritonitis, "and to one who has not come in contact with urachal cyst it is a most pardonable error." The reverse error, however, must seem to many of us as even more probable. A large cyst of the urachus may be mistaken for an ovarian tumour (Bryant, *vide supra*) or for an ectopic gestation sac.

Incision and drainage are the first essentials in *treatment*, and often the operator can do no more.

ALBAN DORAN.

REPORTS OF SOCIETIES.

OBSTETRICAL SOCIETY OF LONDON.

Meeting held January 2nd, 1907, Dr. W. R. Dakin, *President, in the Chair.*

Dr. Archibald Donald read a paper on

Chronic Septic Infection of the Uterus and its Appendages,

which appears *in extenso* on p. 113 of this number of the Journal.

Dr. W. Fletcher Shaw then gave a lantern demonstration, and read a paper on

The Pathology of Chronic Metritis,

which also appears in this number (p. 124).

The President thanked Dr. Shaw for his valuable paper. It was an important step towards putting the study of so-called "metritis" on a scientific basis. Dr. Shaw had carefully controlled his observations on the diseased uteri by examining the uteri of normal subjects, and his resulting opinion was in consequence a safe foundation. The President used the term "so-called metritis" because, as far as he could see, there was nothing indicating inflammation in any of Dr. Shaw's sections or descriptions. In fact, both "metritis" and "endometritis," as used at present by many, did not necessarily mean anything connected with inflammation. These terms, however, led students and others astray, and ought to be reserved for inflammatory processes. He would appeal to both Dr. Donald and Dr. Shaw, as among the pioneers in this research, not to countenance such misuse of terms.

Dr. Cullingworth said that he knew Dr. Donald's paper to be the outcome of much careful clinical observation, of prolonged thought and of strong conviction. The subject was one that had engaged the author's attention to his (the speaker's) knowledge for many years. With reference to Dr. Shaw's paper, he thought the Fellows would be interested to know that it was based upon a thesis which had been presented to the Victoria University of Manchester for the M.D. degree, and to which the authorities of that University had awarded the distinction of a gold medal. The paper represented three years' hard work. The two authors, though both were Manchester men, had not worked in collaboration, but, approaching the subject independently—the one from a clinical, the other from a pathological standpoint, they had gradually become aware of each other's work and of the fact that the conclusions at which they had arrived were practically the same.

Dr. AMAND ROUTH said that no subject in minor gynæcology was more perplexing than that of chronic metritis. Some patients with large hard uteri would suffer from menorrhagia and no local pain, whilst others, with apparently a similar uterus would have no hæmorrhage but considerable pelvic discomfort. Curetting frequently relieves such patients even when the scrapings are insignificant in quantity, when in fact an atrophic "endometritis" has succeeded to the hypertrophic "endometritis" which the authors appear to have mainly dealt with. Did the authors consider all their cases to be "septic" in origin? Were not some of them really of the type usually known as arterio-fibrosis of the uterus.

Dr. RUSSELL ANDREWS said that he thought that Dr. Fletcher Shaw deserved the thanks of the meeting for the clear demonstration of the fact that there were no characteristic histological changes in "chronic metritis." One reason why some gynæcologists were loth to accept chronic metritis as a clinical entity was that the slides and micro-photographs used to illustrate some papers on this subject showed changes in the muscle, connective tissue and vessel walls which were said to be pathognomonic of chronic metritis, though they differed in no way from what was seen in normal multiparous uteri. He thought that this was one of the most interesting points in a very interesting demonstration.

Dr. CUTHBERT LOCKYER expressed his hearty appreciation of Dr. Fletcher Shaw's admirable and painstaking work upon the much vexed question of the pathology of so-called chronic metritis. He thought this Society owed a debt of gratitude to Dr. Shaw for his paper and demonstration which constituted the first really scientific attempt at evolving order out of chaos (as far as this subject was concerned), which had been presented for discussion at the Obstetrical Society of London. It was true that the Society's attention had previously been drawn to certain histological features in uteri said to be in a state of "chronic metritis," but such conditions as were noted could all be found in parous uteri devoid of such clinical symptoms as hæmorrhage and subinvolution. Dr. Shaw's demonstration was convincing from the fact that he was careful to demonstrate the histology of the normal side by side with that of the abnormal uterus, and again his systematic three years' work embraced a mass of material which from its very bulk added valuable weight to his conclusions. It was interesting to note the uniform finding of concomitant mucosal hypertrophy with similar changes in the meso-metrium. In cases the speaker had examined hysterectomy had frequently followed a comparatively recent curettage, and for this reason probably the specimens showed no excess of thickness in the lining membrane of the cavum uteri. Dr. Lockyer was, however, quite familiar with changes in endometrial scrapings which were analogous in all the details of hypertrophy and hyperplasia (including early fibro-blastic invasion and new vessel formation) which are to be found in the fibro-muscular walls of uteri removed for hæmorrhage and increased weight. Dr. Lockyer particularly noted that Dr. Shaw's series of cases did not include one type of uterus in which he himself was especially interested, and to which attention had been drawn by Dr. Goodall at the meeting of the British Medical Association at Toronto in August, 1906, viz., the arterio-sclerotic uterus. The speaker, from his own clinical and pathological experience, agreed with Dr. Goodall that there was a type of uterine disease which affected primarily the vessels of that organ, a disease characterized by great thickening and hyaline degeneration of the middle coats of the vessels together with regular thickening of the intima. These changes

lead to actual occlusion of some of the vessels, to compensate for which new vessels can be found in the process of formation. Now such uteri are found to be associated with general cardio-vascular changes, such as are seen in Bright's disease, and clinically give rise to intractable hæmorrhage. If the latter be treated by curettage the scrapings are *not* abundant, and are principally characterized by a fibro-blastic invasion starting in the deeper layers of the mucosa and working outwards. This leads to fibrous tissue formation, destruction of gland-tubes, and atrophy of the mucosa itself. Attention has been drawn to this condition not only by Dr. Goodall but by Dr. Freeland Barbour in a paper entitled "Climacteric Hæmorrhage due to Sclerosis of the Uterine Vessels" (*Scottish Med. and Surg. Journ.*, June, 1905), and, as this author states, the only remedy for the hæmorrhage is removal of the uterus. Dr. Lockyer cited a similar case from his own practice at St. Mary's Hospital, Plaistow, and it was surprising to him that in such an extensive research Dr. Shaw had not met with this condition. Dr. Lockyer would therefore like to hear from Dr. Shaw whether he regarded every case of so-called chronic metritis as of infective origin, or if he would admit the entity of a primarily arterio-sclerotic uterus occurring independently of any source of infection from without.

Dr. Frank E. Taylor said that he had been much impressed by two points during the routine pathological examination of a considerable number of uteri, similar in character to those described by Dr. Donald and Dr. Shaw, which had been removed on account of excessive and intractable hæmorrhage. The first point was the extreme variability of the histological findings presented by the various specimens examined, some uteri presenting histological features which differed very slightly from those of normal uteri, whilst others presented most striking changes, especially as regards the thickness of the vessel walls in the myometrium. All intermediate stages were likewise observed. The second point was the lack of a normal standard with which to compare the conditions found in chronic metritis—as to what amount of thickening of the vessel walls in the myometrium could be considered pathological and as to the significance of the relative proportion of fibrous to muscular tissues present, along with the difficulty of definitely ascertaining this proportion. He (Dr. Taylor) agreed with Mr. Targett that *a priori* one would expect to find more marked changes in the endometrium than in the myometrium in uteri causing such excessive and persistent hæmorrhage, but in the majority of the cases examined by him the changes in the endometrium, beyond possibly some hyperplasia, were usually not very marked, whilst the myometrium showed more definite changes, which changes usually consisted in great thickening of the vessel walls. It was, he considered, an extremely difficult matter to explain the uterine hæmorrhages on the ground of these histological findings. Both Dr. Donald and Dr. Shaw had laid great stress on the importance of infection as a causative factor in the ætiology of chronic metritis and had mentioned the frequency with which the symptoms dated from a confinement or miscarriage presumably septic. Now gonococcal was probably as frequent as puerperal infection of the pelvic viscera. Hence it might have been expected, if infection were really an important factor, that a history of gonococcal infection would have been present in a certain proportion of the cases, but of such a possible origin neither Dr. Donald nor Dr. Shaw made any mention.

Dr. Donald and Dr. Fletcher Shaw replied.

BRITISH GYNÆCOLOGICAL SOCIETY.

Meeting held December 13th, 1906, Mr. F. Bowreman Jessett, *President, in the Chair.*

Mr. Furneaux Jordan made a further report on a speciman exhibited at the last meeting, and stated that it was an œdematous fibroid.

Dr. Swanton read notes on a case of *Abdominal Hysterectomy for Fibroid under Spinal Analgesia,* and showed the specimen. The tumour, which weighed three and a half pounds, was removed from a patient, aged 44, complaining of hæmorrhage and pressure symptoms. It was removed by a modification of Doyen's method. The intra-spinal injection consisted of 6 c.c. of a 5 per cent. isotonic solution of novocain and suprarenin. The tumour was removed without any pain or shock, but a general anæsthetic was used while passing some of the final sutures. The patient left the hospital on the twenty-fourth day.

Mr. Charles Ryall showed a *Uterus removed under Spinal Analgesia for Hyperplastic Endometritis.* The patient was aged 30, and complained of menorrhagia; she had been curetted several times with an unsatisfactory result. The uterus was large and cervix patulous. Hysterectomy was decided on, and owing to her exhausted condition it was performed under spinal analgesia. She was injected with 8 c.c. of novocain and suprarenin solution. Analgesia followed in ten minutes, and the entire operation lasted forty-one minutes. The patient did not complain of actual pain, but when the uterus was seized and drawn up she was conscious of something being pulled on. The operation was satisfactorily concluded, and the patient made an uninterrupted recovery. He considered spinal analgesia was preferable in some classes of cases, such as the condition known as "acute abdomen," especially in its later stage; also in acute or serious chronic pulmonary lesions and in certain cardiac diseases. In major operations he considered it an advantage to have a patient unconscious. Shock was hard to be diminished under spinal analgesia, but he considered that by careful manipulation of the abdominal viscera it could be lessened to a large extent.

Dr. Hodgson read a paper on

A Cause of Fatal Collapse after Prolonged Abdominal Operations.

The selection of an anæsthetic by the operator was a moral obligation, and an important one from a patient's point of view. Fatal collapse was often attributed to the length of time occupied by the operation rather than the unsuitability of the anæsthetic. Each case ought to be considered on its own merits and the anæsthetic chosen accordingly. More deaths follow long operations on the abdomen when ether is used than when chloroform is employed. The effects of ether and chloroform were contrasted in their action on the lungs and heart. The opinion was expressed that both these anæsthetics produce their effect by impeding the proper elimination of poisonous substances. Fatal cases arise from causes outside the patient and often depend either upon the anæsthetist or the operator, and so far are preventable. Both anæsthesia and operation are inseparable from the operator.

Dr. Macnaughton-Jones read *Notes from the Clinics of Heidelberg and Freiburg,* illustrated by the epidiascope. The paper gave an account of interviews with Prof. v. Rosthorn at Heidelberg and Prof. Kroenig at Freiburg; also observations upon technique and methods of treatment.

GLASGOW OBSTETRICAL AND GYNÆCOLOGICAL SOCIETY.

Meeting held November 28th, 1906, Dr. OLIPHANT, *President, in the Chair.*

FRESH SPECIMENS SHOWN.

Dr. OLIPHANT:

1. *Adeno-Carcinoma of the Ovary*—secondary degeneration in a benign cyst.

2. *Fibroid Tumour of Uterus.*

3. *Cystic Fibroid of the Ovary.*

4. *Hydrosalpinx and Cyst of the Ovary.*

Dr. MUNRO KERR:

1. *Extra-Uterine Pregnancy,* in which rupture occurred first into the broad ligament, and then two months later into the peritoneal cavity.

2. *Ruptured Extra-Uterine Pregnancy at about Six Weeks' duration.* The other tube showed a hæmatosalpinx.

3. *Cyst of Ovary* with diverticula on tube filled with blood.

4. *Fibroid Tumour of Left Broad Ligament.*

Dr. JOHN LINDSAY:

Placenta from Twin Pregnancy with two cords in actual contact at origin, twisted round one another and tied in a knot. The twins were dead-born and macerated.

Dr. WILSON:

Microscopic Sections of Epithelioma of Vaginal Walls (these were referred to the Pathologist for a more detailed report).

Dr. JARDINE read a paper on

SOME INTERESTING CASES OF PREGNANCY AND LABOUR,

which he divided into four groups:—

GROUP I. *A Case of Premature Labour in a Paraplegic Patient.* Labour came on at the beginning of the seventh month, the child being born dead. The patient had complained of paralysis of both legs for four years, due to an accident while boating. On examination of patient at the time of confinement it was found that there was present atrophy of the muscles of the lower limbs, ankle clonus, exaggeration of the knee-jerks with difficulty in straightening out the legs. The labour was easy and the puerperium was normal.

GROUP II. *Cæsarean Section for Contraction at the outlet of the Pelvis.* Patient i.-para, pregnant eight months at onset of labour. The pelvic outlet was much contracted, the distance between the ischial tuberosities being 1½ inches. Cæsarean section was performed, and patient and child did well.

GROUP III. *Three Cases in which there was difficulty in delivering the Body after the Head had been Born.* In one craniotomy was performed owing to the narrow condition of the pelvis. After the head had been entirely separated from the body one arm was removed before delivery of the remaining portion of the child could be effected. In the second case the child was dead on account of prolapse of the cord; craniotomy was performed, and the difficulty in the delivery of the shoulders was found to

have been due to a marked contraction ring in the uterine wall. In the third case the head was delivered by forceps, but as the shoulders were impacted the patient was sent into hospital. Traction was made upon the arms and delivery effected, the delay being due to the large size of the child.

Group iv. *Toxæmia of Pregnancy.* The patient, 7½ months pregnant, became unconscious for a short time while at her work. Paresis of the left side was then observed. The urine was loaded with albumen and was scanty in quantity. The pupils showed irregularity. After treatment for two weeks labour was induced and ended naturally by the birth of a living child. The patient made a good recovery, but seemed somewhat abnormal in her mental condition.

Dr. Jardine's paper was discussed by Drs. Kelly, Munro Kerr, Carstairs Douglas, Balfour Marshall and the President.

Dr. Jardine replied.

EDINBURGH OBSTETRICAL SOCIETY.

Meeting held Wednesday, December 12th, 1906, Dr. N. T. Brewis *in the Chair.*

Dr. Brewis read a communication on Two Cases of Pregnancy complicated by Fibroid Tumours and Treated by Hysterectomy. In the first patient the tumour grew from the posterior aspect of the supra-vaginal portion of the cervix; it filled up the pelvis and was palpable in the abdomen to the left of the uterus. Interference became necessary at the fifth month owing to severe and persistent pain, threatened intestinal obstruction and bladder troubles from urethral pressure. The fœtus was delivered by Cæsarean section and the tumour and uterus were removed by supra-vaginal hysterectomy. In the second patient the tumour was situated to the left of the uterus just above the pelvic brim. The case was allowed to go to term, and at the onset of labour Cæsarean section was performed. Owing to the condition of the base of the tumour and the presence of another fibroid in connection with the body of the uterus, it was found to be advisable to remove the uterus by supra-vaginal hysterectomy. In discussing the treatment of pregnancy complicated by fibroid tumours, Dr. Brewis emphasized the importance of the site of the growth in determining the means to be employed.

The paper was discussed by Drs. Ritchie, Lamond Lackie and Keppie Paterson.

Dr. Haig Ferguson contributed a Note of a Case of Complicated Labour in a Primipara with One Kidney, which will appear as an original paper in a subsequent number of the Journal.

Drs. Brewis, Ritchie and Porter spoke on the subject.

A paper on Successful Treatment of Puerperal Fever by Anti-streptococcic Serum, by Dr. Garnet Leary, was read by the Secretary.

Specimens.

Dr. Brewis showed (*a*) *Two Examples of Endothelium of the Ovary* removed from a patient aged 20; (*b*) *Rare Variety of Dermoid Tumour of the Right Ovary, Tuberculous Tubes and Adenomatous Tumour of the Left Ovary* removed from the same patient; (*c*) *A rapidly-growing Dermoid*

Tumour which ruptured during administration of the anæsthetic; death on the third day probably due to absorption of the fatty material extravasated; (*d*) *Ruptured Ovarian Tumour* presenting microscopic characters of adeno-carcinoma and tubercle; (*e*) *Uterus with large Cervical Fibroid* removed at the fifth month of pregnancy; (*f*) *Uterus with Fibroid Tumour in Lower Uterine Segment* removed at term after Cæsarean section.

Dr. HAIG FERGUSON showed (*a*) *Two Cases of Tubal Pregnancy*; (*b*) *Large Pyosalpinx*; (*c*) *Uterus with Fundal Cancer* removed by vaginal hysterectomy; (*d*) *Chorion-epithelioma of Uterus* removed by vaginal hysterectomy; (*e*) *Inverted Uterus*; the condition had come on insidiously after labour, and when recognised was found to be irreducible; (*f*) *Soft Subperitoneal Fibroid* resembling ovarian cyst; (*g*) *Chart* showing antepartum temperature of 105·8°F. (malarial), where child was born alive; (*h*) *Uterus* removed on account of persistent foul discharge from a patient who had undergone double salpingo-oöphorectomy a year previously.

Dr. RITCHIE showed a very complete *Early Ovum*.

Dr. HAULTAIN showed a curious *Tumour*, about the size of a placenta expelled before the child during labour in a multipara. It was found to consist chiefly of soft vascular tissue with a small denser necrotic area. The cord and placenta were normal. No explanation was offered as to its nature and origin.

REVIEWS OF RECENT BOOKS.

THE DEVELOPMENT OF THE HUMAN BODY: A Manual of Embryology. By J. Playfair McMurrich, A.M., Ph.D., Professor of Anatomy in the University of Michigan. Second edition. Pp. 560; 272 illustrations. London: Rebman Limited, 1906. Price 14s. net.

It is but three years since the first edition of this text-book on human embryology appeared. The second edition just published embodies many modifications, which have been introduced as a result of recent contributions to our knowledge of the organogeny of the mammalia. In a text-book which so well describes the facts of human development this modernization is of the utmost value to the student of embryology. Frequent revision is more especially necessary in this subject because of the many blanks and breaks in continuity which still exist in our knowledge of it. The author is to be congratulated on having produced this second edition with the addition of but eleven pages of text and two illustrations.

The book as brought up to date comprises a little more than 500 pages in which are 272 illustrations. After a careful perusal one instinctively feels that the author has performed his task with remarkable fairness to all. The opinions of contending embryologists are described with complete impartiality. The book paints the picture of human development as far as we know it with clearness and continuity.

The opening chapters describe in considerable detail the development of spermatozoa, ova and the great primitive germ layers. In starting with this great primary developmental groundwork, the book compares very favourably with other well-known text-books on the subject which carry us at once into the evolvement of one or other of the great systems, such, for example, as the special senses. It is impossible of course to tell the romantic story of animal growth with anything like chronological sequence, but a description of the initial stages is essential in order to understand how the great systems arise.

This text-book in its opening chapters presents very clearly to the student that essential basement upon which his imagination can place the succeeding structure of systemic development.

Certain chapters are written with great clearness and success. Especially is this the case in those describing the yolk sac, the belly stalk, the urogenital, osseous and muscular systems, as well as certain parts of the alimentary and circulatory systems. The development of the fœtal membranes, though described with lucidity, lacks the detail which a more advanced student would desire. The well-known views, for example, of Berry Hart on the formation of the amnion as well as his interpretation of the development of the vagina are not mentioned. It is, however, necessary to remember that the book has been written primarily for the student, and thus many points of detail interesting and important to the special scholar must be excluded.

Many of the schematic tables dealing with such matters as branchial arch development, cranial development, homologic structures in male and female genital development, and others are calculated to help the student in mastering and remembering the gradual evolvement of the many structures involved.

The text-book is printed in large clear type. The author's style, though generally clear and intelligible is not at all times free from obscurity. This, however, is largely due to our imperfect information, and to the necessity of compressing the matter within a limited space. The illustrations are the least successful part of the book. A large number of them are excellent, but many lack precision and meaning. There is appended to each chapter a short bibliography dealing with the subject under discussion.

It is quite certain that this book must remain for many years one of the best English text-books for students of human embryology.

THE TREATMENT OF SYPHILIS, pp. xvi. and 276. THE PROPHYLAXIS OF SYPHILIS, pp. xii. and 220. Large 8vo. in one volume. By Alfred Fournier, Professor at the Faculty of Medicine; Physician to the St. Louis Hospital, Paris. English translation of the second edition (revised and enlarged) by C. F. Marshall, M.D., F.R.C.S., late Assistant Surgeon to the Hospital for Diseases of the Skin, Blackfriars, London; formerly Medical Officer to the London Lock Hospital. London and New York: Rebman Limited. Price 21s. net.

Professor Fournier has made syphilis almost a lifelong study. This fact and the unique position which he holds as an authority on this disease suffice to make any writing of his on this subject sure to be looked forward to as something likely to be both interesting and valuable, and in this expectation the reader of the volume before us has no reason to be disappointed.

Amongst the many questions to which the author draws attention we are glad to see the inadequate, if not shameful, position of the teaching on the treatment of syphilis in our medical schools at the present time. He regrets that no attempt has yet been made to make the treatment of such a serious disease an important part of the student's curriculum. The student is taught how to deal with various syphilitic lesions, but too little importance is attached to the necessity of treating the disease itself with thoroughness and perseverance. We regret that we in England can corroborate the truth of this accusation.

Professor Fournier does not believe that there is such a thing as benign, or a mild attack of, syphilis, and he emphasizes the fact that the disease, no matter how mild apparently, should be treated with the same seriousness and for as long a time as if it were an obviously severe attack. In other words, all cases must be treated with the same rigour.

The fallacy of abortive treatment and the uselessness of excision of the chancre are disposed of in a convincing manner.

The general treatment of the disease is dealt with exhaustively, with special reference to the details of the many various methods of administering mercury, and we would draw special attention to the valuable information contained in the chapters on inunction, and the various methods of carrying out treatment by injections into the tissues.

It is satisfactory to note the author's conviction that as to the administration of mercury, preference should be given to ingestion, the results of which have generally been found so satisfactory in this country. He gives very valuable information on the use of iodides, and as to how and when one should resort to these remedies.

Another important part of treatment is hygiene, not only with regard to the patient's care of himself, but also for the protection of others.

The factor of syphilis in pregnancy is a subject of much significance to the obstetrician, and Fournier attaches great importance to this question. Repeated abortion, or death of the fœtus he believes to be generally due to this disease, hence the necessity of enquiring into the possibility of past infection in the husband or wife, and treating either or both when there is reason to believe that such has occurred. He gives many instances of the satisfactory results following such treatment.

In the second part of the work, viz., "The Prophylaxis of Syphilis," he goes over a good deal of ground on the debatable subject of the attitude which the State ought to take with regard to that unfortunate class by whom the disease is mainly propagated. A perusal of his views on the subject ought to be convincing to those in this country who have taken up a let-things-alone attitude towards this question, and who still shut their eyes to the necessity for some amendment of the Contagious Diseases Act. As a step towards prophylaxis a great deal can be done by educating the public on the danger and after effects of the disease, and it is our duty to do so, and not to shirk our responsibilities in avoiding what is an unpleasant question.

The effect of syphilis on infant mortality is undoubtedly a matter of extreme gravity, and the author produces some startling figures on this subject which almost lead one to believe that he views this question with undue pessimism. He shows what a serious factor it is in the causation of depopulation, especially in France.

The relation of the wet-nurse to syphilitic sucklings, and under what conditions we should allow or refuse a wet-nurse to the child who has a syphilitic father, the danger, in using human instead of animal vaccine, of producing vaccinal syphilis, and the preventive treatment of paternal syphilitic heredity during pregnancy, are all of them important questions and are all discussed in this volume.

The work consists of two parts bound in one volume. It is full of interest and valuable instruction, and forms an excellent work of reference on questions pertaining to the general treatment of syphilis.

Much praise is due to the translator, who seems to have grasped the true spirit of the author, with the result that the reading of the book is both easy and pleasant. It is one of the best translated works that we have come across.

C. R.

THE PUERPERIUM. By C. Nepean Longridge, M.D., F.R.C.S. London: Adlard and Son. Price 5s.

A trustworthy guide to the management of the puerperium, which at the same time shall be concise and practical, has been much wanted. The larger text-books devoted to obstetrics deal with this important phase of the obstetrician's duties in a more or less academic style—scientific but

somewhat unpractical. In the smaller works the authors, from lack of space, are compelled to utilize the bulk of their pages to describe and explain those essential facts on which the whole structure of midwifery is built. Thus it comes about that many students and young practitioners lack that intimate knowledge of the emergencies and every-day complications of the puerperium which is so requisite for successful practice.

Both these classes of readers owe a debt of gratitude to Dr. Longridge for setting forth in an eminently readable form the practice and teaching of the premier lying-in hospital in London. The book is indeed entirely founded on the work done at Queen Charlotte's Hospital, and represents a wealth of practical hints for those whose experience of the lying-in woman and new-born child is just beginning.

The subjects of post partum collapse and hæmorrhage are admirably dealt with. The section devoted to puerperal sepsis, its prevention and treatment, is practical and full of sound common sense, whilst the minor sequelæ of labour are discussed in no less thorough a manner.

The chapters devoted to the management of the infant are admirable and their teaching not to be gainsaid. The full directions given in regard to infant feeding cannot but prove useful to those confronted in practice with the troublesome gastro-intestinal disorders of infancy.

The author's style is very distinctive, and makes subjects ordinarily dry not only interesting but often amusing, so that the book can really be read with pleasure.

Indeed the only fault we can find is a certain exuberance of metaphor which occasionally shows itself in the analogies with which the author enforces his points. In the second edition, which we have no doubt will shortly be wanted, these might be subjected to a little judicious pruning.

V. B.